The Journal of Thoracic Surgery

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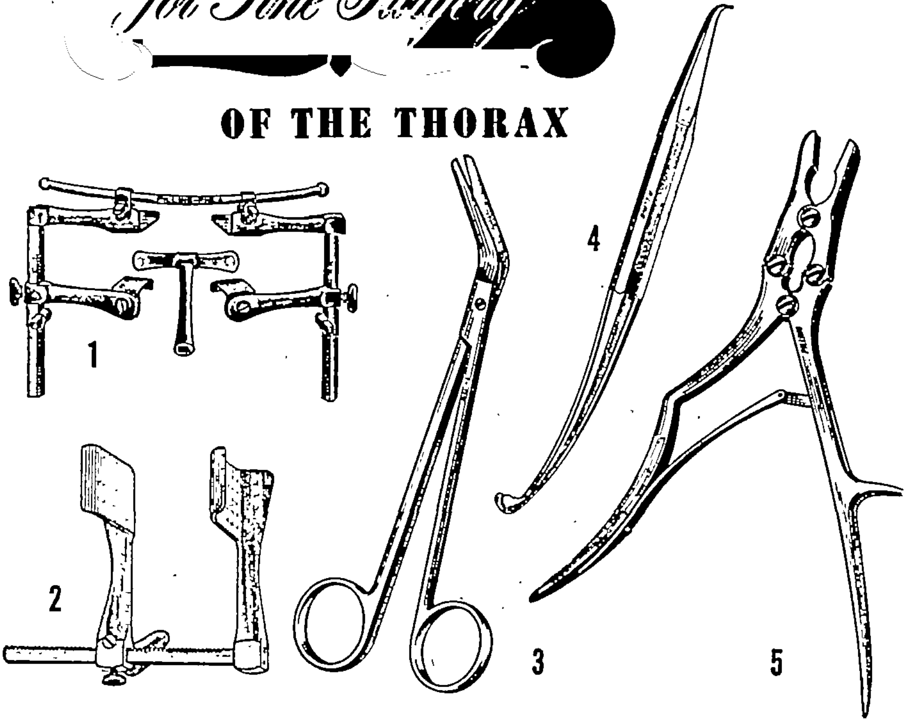

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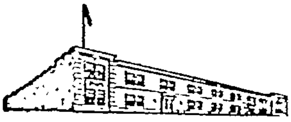

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The Journal of
Thoracic Surgery

Vol. 16 December, 1947 No. 6

Original Communications

DECORTICATION IN CHRONIC EMPYEMA OF TUBERCULOUS ORIGIN

Fraser B. Gurd, M.D., C.M.
Montreal, Que.

During the years 1919 to 1925, approximately seventy patients with chronic empyema were admitted to my service at Ste. Anne's Military Hospital. This comparatively large number of patients were soldiers and sailors of the Canadian services. They had developed chronic empyema in consequence of infected pleural cavities as the result of both gunshot wounds and as a sequel of the pandemic of influenza. In June, 1921, the results of treatment in the first group of twenty-five patients were reported.[1] Since the results in the whole group of seventy patients were in every way comparable to those reported, a short analysis of the smaller group is included here.

Of the twenty-five referred to above, in twelve the condition followed gunshot wounds, in twelve it was the result of empyema, and in one it was tuberculous in origin. In ten cases of smaller cavities, cure followed improved drainage or removal of foreign bodies. One patient left the hospital improved but not healed. In this group referred to as smaller cavities, the average duration of disease was thirteen months, the longest twenty-four months, the shortest six months.

In the group of large cavities, in six of which there was exhibited either complete or almost complete collapse of the lung, decortication was carried out in ten. In two of these immediate expansion of the lung failed to take place. In one of these two, the chest wall was collapsed with ultimate cure. In the other, a soldier whose condition was *in extremis* upon admission and who suffered from nephritis, a limited decortication was attempted. No expansion of the lung occurred and no further operative interference was attempted. This was the only death in the series. In this latter group of fourteen patients, the length of time during which the disease had lasted prior to admission to Ste. Anne's Military Hospital was from eight to fifty-four months.

From the Montreal General Hospital, the Royal Edward-Laurentian Hospital, and the Grace Dart Home Hospital, McGill University, Montreal.

Read at the Twenty-Sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

At the meeting of the Association in Boston, in 1921, a paper entitled "Scoliosis accompanying Chronic Infected Open Pneumothorax" was read.[2]

Reference is made to this article at this time because the history and accompanying pictures illustrated the extreme deformity which may occur in a young person as the result of long-standing empyema.

During the years immediately following the last war, a number of contributions upon the subject of chronic empyema were published on this continent, notably those of Keller, Hedblom, and Eggers. In all three of these articles, reference was made to decortication and, in the main, the results reported from the performance of this procedure were satisfactory, although not enthusiastic.

Although very little reference to chronic empyema has been made before this Association, or, for that matter, elsewhere, over a period of many years, in 1941 the subject of chronic nontuberculous empyema was presented by Singleton and Moore[3] and was followed by a very general discussion in which Bigger, Eggers, Carr, and others, including that great gentleman, Howard Lilienthal, took part. The general tenor of this contribution and of the discussion was to support decortication of the lung in the treatment of certain carefully selected cases of chronic empyema. The fact that the procedure preserves pulmonary function and avoids marked deformity, as compared with chest wall collapse, was stressed by all speakers. Of special interest in so far as my contribution today is concerned is the fact that, among the contra-indications to decortication, tuberculosis was referred to by all speakers as perhaps the most important.

It is fully realized by the author that the study of three cases in which operation for chronic empyema complicating pulmonary tuberculosis has been performed is not proof that the procedure which is being suggested has any wide applicability, nevertheless, in view of the relative frequency of the disease, in view of the unsatisfactory results obtained by other methods, including extrapleural thoracoplasty and operations of the Schede type, and in view of the crippling deformity which follows in those who survive extensive operations for collapse of the chest wall, I believe that the matter of decortication deserves consideration. I make, therefore, no apology for basing a discussion on such a small number of cases.

In consequence of early experience in decortication for chronic empyema, I have continued in the intervening years to perform this operation on a moderately large number of patients every year. Since the results have been, in the main, satisfactory and unfavorable complications or sequelae are substantially absent, I undertook an experiment two years ago of performing decortication of the lung in a patient with chronic empyema due to tuberculosis. Since that time, two other patients with chronic empyema which complicated artificial pneumothorax in the treatment of pulmonary tuberculosis have been operated upon.

Prior to 1940, in our clinics at both the Grace Dart Home Hospital and the Royal Edward-Laurentian Hospital, the incidence of empyema complicating pneumothorax had been distressingly high.[4] Fortunately, since that date, in consequence of better realization on the part of physicians in our community of the risk of an inadequate pneumothorax, the number of patients with this serious complication has very materially diminished. We have had a large number of these patients under our care in the past, however, and have been impressed by the unsatisfactory effect of extensive thoracoplasty of either the extrapleural or Schede types.

During the summer of 1944, a child 9 years of age, R. McM., was admitted to the Royal Edward-Laurentian Hospital, Montreal Division, having suffered from a mixed tuberculous empyema for over twelve months, for which thoracotomy had been performed. Since it was realized that serious deformity was almost inevitable in a child of this age whether the disease could be arrested by more adequate drainage alone or by extensive thoracoplasty, it was decided to attempt decortication. This decision was arrived at despite the overwhelming consensus of opinion that tuberculosis constituted a definite contraindication to the procedure, and despite the fact that no one in our group had had any experience in the performance of the operation for tuberculous disease.

R. McM. was first seen in July 1941, at the age of 6. He belonged to a family many of whom were suffering from pulmonary tuberculosis. Although Mantoux reaction was positive, no definite evidence of pulmonary disease was noted until August, 1943. On this latter date he was admitted to the Royal Edward-Laurentian Hospital suffering from a total left-sided empyema with displacement of the mediastinum and mixed culture of Type VI pneumococci, and *Staphylococcus pyogenes* was isolated. Rib resection for drainage was carried out with improvement in the child's condition, which had been very grave.

The child's condition improved as the result of drainage, but two months later a heavy growth of tubercle bacilli was obtained. In the interval, he had been transferred to the Children's Memorial Hospital. In January, 1944, he was re-admitted to the Royal Edward-Laurentian Hospital and a preliminary extensive thoracotomy was carried out. The wound was left wide open and packed with liquid paraffin "bipped" gauze. As the result of this more adequate drainage, a substantial improvement in his condition occurred. The thoracotomy incision was further enlarged so that the whole base of the cavity was clearly exposed, and in August, 1944, that is to say, twelve months after the onset of the empyema, decortication was carried out.

At operation it was noted that the parietal pleura had thickened to about 1.5 cm. and the ribs were overlying one another. The membrane overlying the lung, pericardium, and diaphragm varied in thickness from 2.5 to 3 mm. and showed in the gross many tubercles. Decortication was carried out without unusual difficulty, although, as has been my experience in most cases of chronic empyema, small areas of damage to the lung surface were inflicted. Air bubbles appeared at different spots. Hemorrhage was insignificant and was easily controlled by packing. After the lung surface forming the bases of the cavity had been freed from dense, fibrous, tissue membrane, it was dissected free from the mediastinum anteriorly and from the vertebral gutter posteriorly. It was also carefully separated from the medial portion of the diaphragm. In this way no great difficulty was experienced in separating the small mass of lung tissue up to the hilar region.

An attempt was made to expand the lung by means of increased pressure through the intratracheal tube through which cyclopropane had been administered. This procedure, although it caused slight expansion of the lung, was not effective; the child was, therefore, allowed partially to regain consciousness and coughing was induced. As soon as he coughed the lung ballooned out and became pale pinkish and downy. As coughing continued, herniation of the lung through the large thoracotomy wound occurred.

Two thicknesses of soft gauze which had been soaked in liquid paraffin to which a small amount of "bipp" had been added were carefully laid over the surface of the expanded lung so that "curtain drainage" was established between the lung surface, which, as has been previously pointed out, was damaged over many small areas, and the interior of the thoracic cage. This curtain of gauze was brought out through the lower part of the wound, which was elsewhere closed with through-and-through silkworm gut sutures.

Despite the fact that the child's general condition was very grave, he recovered promptly from operation and on Oct. 16, 1944, he was discharged, general condition much improved, sputum negative, and wound healed except over the lower part where an ulcer about 4 cm. in diameter remained. Two months later he was readmitted to hospital and the somewhat saucer-shaped, granulating, tuberculous surface over the lower part of the wound was excised. The skin edges were undercut and the wound partially closed by suture. He was again returned home and again readmitted for the excision of a small remaining tuberculous ulcer. Complete healing of the chest wall was the result, and he was discharged to the Julius Richardson Convalescent Hospital (for children) for the summer. During his stay in the country he improved enormously and in October he returned to school.

He was re-examined April 26, 1946. The chest wall is soundly healed and he has again commenced to grow. Examination of the chest shows that aeration of the left lung is being accomplished. The accompanying illustrations of x-ray examinations (Fig. 1) indicate, I believe, the progress of this little fellow's state over a three-year period. I believe that it is reasonable to argue that he has been salvaged not only in so far as life is concerned, but that it will be possible for him to grow up as a reasonably useful citizen.

R. McM. was, therefore, the first case in which decortication of the lung for chronic tuberculous empyema was carried out. The indications for attempting the procedure we considered sufficient, and happily the result to date seems to indicate that decortication for tuberculosis is possible, and that it may be satisfactory in so far as, (1) arrest of disease, (2) avoidance of crippling deformity, and (3) at least partial return of pulmonary function, is concerned.

Not many months after R. McM. had been operated upon, a young woman, who had developed a complete right-sided empyema during the course of treatment of extensive apical disease by artificial pneumothorax, was brought before Conference at the Royal Edward-Laurentian Hospital. Since no bacilli were present in the sputum, she was transferred to the Montreal General Hospital and decortication was carried out. Her case history follows.

H. M., 24 years old, was admitted in December, 1941, to the Royal Edward-Laurentian Hospital with moderately advanced right-sided apical tuberculosis with cavitation. In January, 1942, pneumothorax was commenced, and in April, 1942, a pneumonolysis (endoscopic) was accomplished. The patient's cough and sputum ceased and tubercle bacilli could not be found.

Pneumothorax was maintained and she was an ambulant patient until December, 1944, when she suffered a sudden onset of pain in the right chest and fever. She was admitted to another hospital and, under aspiration, thick, purulent material was discovered which showed a culture of *Staphylococcus aureus*.

In April, 1945, she was again admitted to the Royal Edward-Laurentian Hospital. She was running a septic type of temperature and looked extremely ill. She did not suffer from cough and expectorated no sputum. Repeated aspirations of the cavity were carried out and penicillin was introduced. By May 30 there were no microorganisms in the aspirated fluid, as shown by smear. Her fever subsided and her general condition improved.

On June 14, 1945, she was transferred to my service at the Montreal General Hospital. Aspiration and penicillin were continued. Transfusions were administered, and on July 3, 1945, an intercostal catheter was introduced and underwater negative pressure drainage

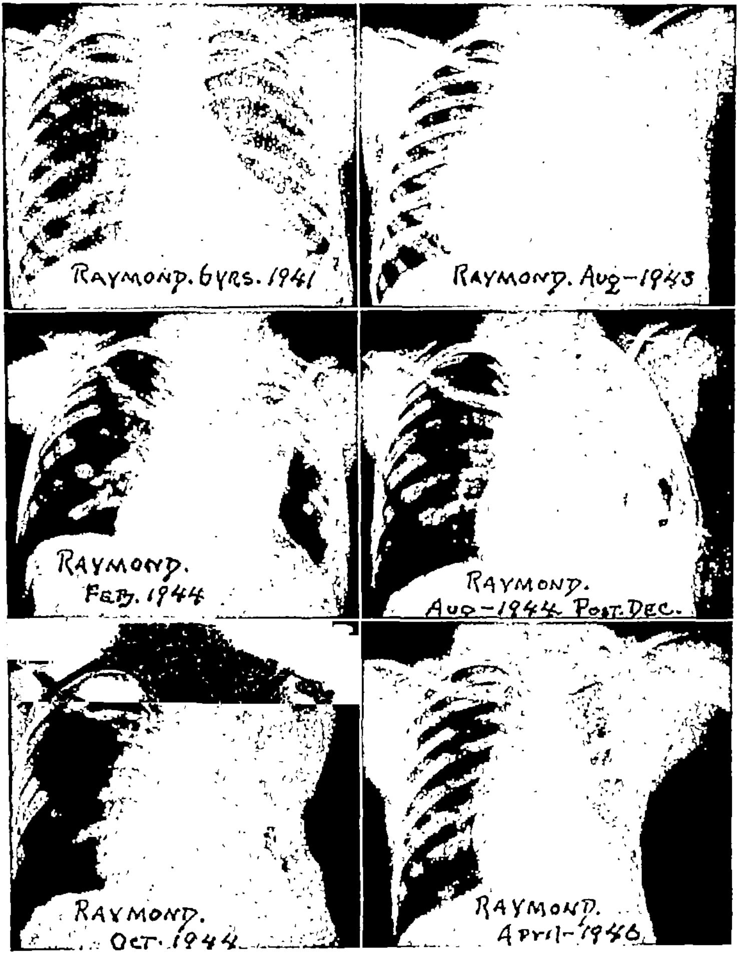

Fig. 1.—A series of six dated photographs which show progress in the case of R. McM.

established. Daily instillations of 100,000 units of penicillin were continued. This form of negative pressure draining resulted in but slight change in the size of the cavity.

On August 1, and again on August 7, thoracotomy was carried out. The sixth, seventh, eighth, and ninth ribs, together with the underlying parietal pleura and intercostal bundles, were removed in order that a sufficiently large opening in the chest wall might be made so that decortication might be attempted.

On Aug. 31, 1945, decortication of the right lung was carried out. The parietal pleura, as previously noted, and as indicated in the x-rays, was found to be nearly 2 cm. in thickness. The cavity was dry. The removal of the cortical membrane, which was slightly less than 3 mm. in thickness, was accomplished without any considerable difficulty and with but slight damage to the lung surface. The adhesions between the collapsed lung and the mediastinum were divided without any great difficulty. The mediastinum had been displaced to such an extent that the prominences of the vertebral bodies were in the left half of the chest. In other words, the dead space occupied somewhat more than a complete hemithorax.

Operation was carried out under intratracheal cyclopropane anesthesia and, although an effort was made to inflate the lung by increasing the pressure from within, this proved to be of little value; the patient was, therefore, permitted to come out of the anesthetic so that she might cough. Immediately the lung became downy throughout and completely filled the chest.

It should be noted that over the uppermost part of the cavity no attempt was made to remove the membrane, since this had recently been the site of an active tuberculous process; the area left undisturbed, however, was small. A self-retaining catheter was inserted and negative pressure drainage carried out for two weeks. The wound healed per primam, and the young woman's general condition improved rapidly. She was discharged from the hospital November 16 and was last re-examined April 26, at which time she reported that she had recently been fitted for her wedding gown.

On Oct. 10, 1945, there was admitted to my service at the Montreal General Hospital, J. Mc., 36 years of age. His history indicated that he had suffered from pulmonary tuberculosis since April, 1938. Pneumothorax had been induced and continued until November, 1944, although fixation of the apex had been present throughout this period. In March, 1945, empyema developed which completely filled the left chest and displaced the mediastinum. Intercostal drainage had been instituted in June, 1945.

At the time of admission there was no evidence of parenchymatous disease. He did not suffer from cough nor expectoration. There were no tubercle bacilli in either sputum or gastric content. Exudation from the chest cavity showed a mixed infection but no tubercle bacilli.

It was decided to attempt decortication of at least the lower lobe. In order that the disease in the upper lobe might not be reactivated if possible, a typical upper-stage extrapleural thoracoplasty was carried out October 18, the upper three ribs being removed in toto, together with the transverse processes of the third and second thoracic vertebrae. This procedure was carried out under spinal anesthesia.

On November 12, a first-stage thoracotomy was carried out under cyclopropane anesthesia, portions of the ninth and eighth ribs being removed. Three weeks later second-stage thoracotomy was done, and portions of the seventh and sixth ribs were resected, together with parietal pleura and intercostal bundles. Following both these procedures the wound in the chest wall was left open and filled with gauze, in the first instance impregnated with sulfathiazol emulsion, and, in the second, with liquid paraffin and bismuth-iodoform-paraffin paste.

On Jan. 4, 1946, decortication was carried out under cyclopropane anesthesia (intratracheal). Although the expansion of the lung was less marked than was hoped for, it seemed as though obliteration of the pleural space would be obtained. An intercostal catheter was introduced for negative pressure drainage; this, however, was removed too soon, and a purulent exudate reaccumulated, with the result that collapse of the lung reoccurred.

Since March 19, three further collapsing procedures upon the chest wall have been carried out with obliteration of the cavity and apparent cure. These procedures have been

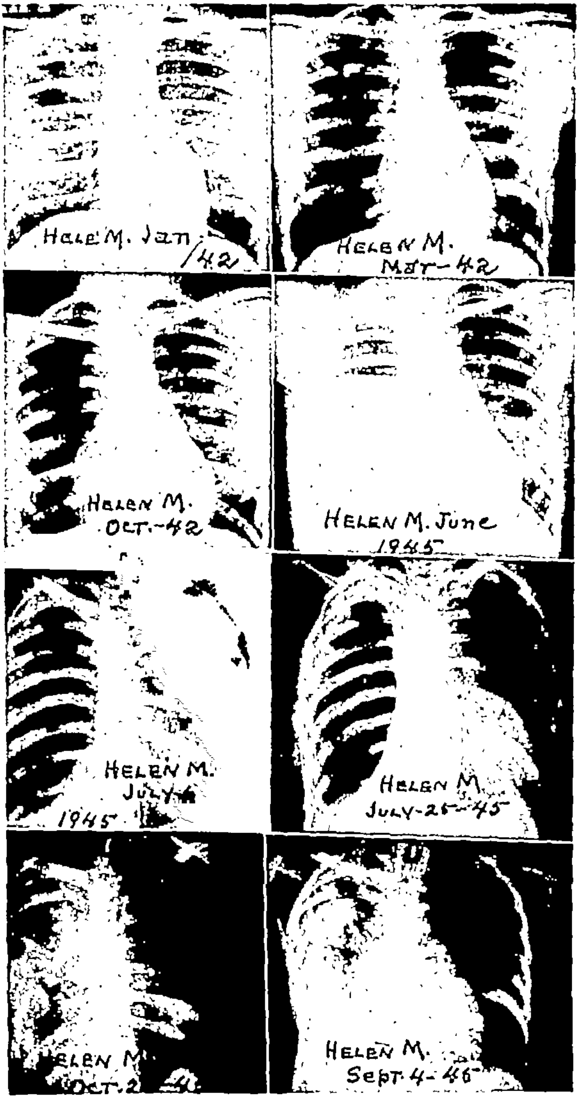

Fig. 2.—A series of eight dated photographs which show progress in the case of H. M.

of the Schede type and have been characterized by the removal of the lower two-thirds of the scapula. The patient's general condition has remained excellent and at no time has there been any anxiety regarding his state.

This case is included in this report since although the result of decortication was disappointing, the procedure has been carried out without any considerable difficulty and the patient suffered no damage as a result of operation. Failure to obliterate the cavity by the reexpanded lung was due, I believe, to the fact that the chest wall was allowed to close prematurely and fluid (pus) to reaccumulate. In any event, the cavity was reduced to approximately one-half its previous size, so that radical thoracoplasty was more effective.

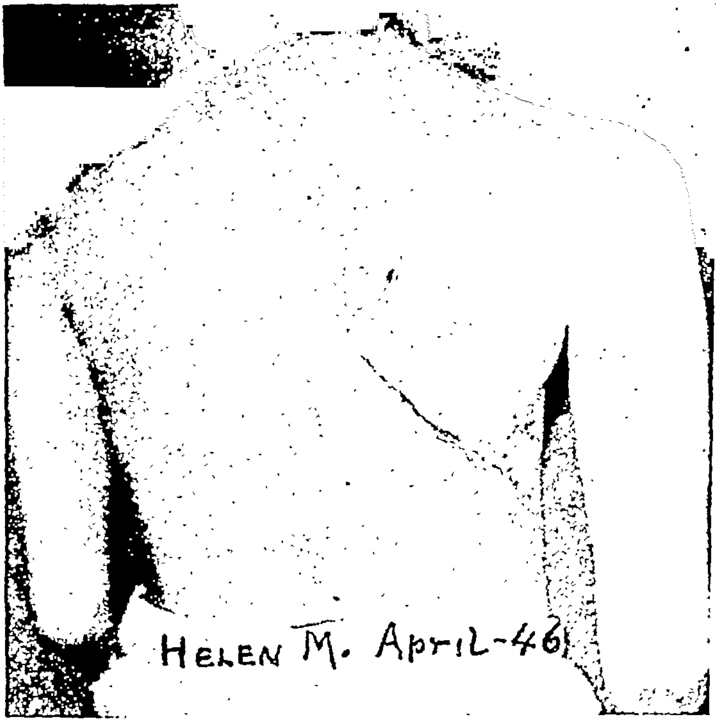

Fig. 3.—Photograph of H. M. nine months after decortication.

TECHNIQUE

Since the author has had an unusual experience in the operation of decortication over a period of many years, it seems justifiable to indicate the technique found to be, on the one hand, safe, and, on the other hand, relatively satisfactory. It is unnecessary to suggest that in view of the fact that many of these cases are exhausted by prolonged suppuration and so illustrate the phenomena associated with anemia and hyproproteinemia, adequate efforts should be made to overcome this state. The procedures available for this purpose will not be referred to in this contribution.

In view of the fact that one of the common causes of chronic empyema is inadequate drainage, this should obviously be provided for. The exhibition of bacteriostatic drugs is, of course, indicated.

Since, in all cases of chronic empyema, including those due to tuberculosis, the parietal pleura is thickened so that the natural elasticity of the chest wall is lost, and since the contractile processes in the thickened pleura result in close approximation of even overlapping of the ribs, the usual method of thoracotomy by means of rib spreaders is not applicable. In order that removal of the dense, fibrous membrane from the surface of the lung may be carried out without hazard, it is necessary that a sufficiently large window in the chest wall be established. Since but few of these cases are good surgical risks, it is advisable to make the opening into the chest in stages, two or three as the case may be. An adequate window having been thus produced, the patient is prepared for operation, and blood in adequate amounts is made available.

The choice of anesthetic will depend in part upon the custom of both the operator and the anesthetist. Since the surface of the chest wall is heavily infected, we have been loath to employ spinal anesthesia; we have, therefore, used either intratracheal cyclopropane or nitrous oxide-oxygen with or without ether, or local anesthesia. The main reason, perhaps, for the employment of intratracheal anesthesia is in order that adequate clearance of the bronchial mechanisms may be established, since there is always the risk that blood may enter the lung from the cut surface, although this complication has not been observed.

In the chronic case which has lasted for six to twelve months or longer, it may be expected that the parietal pleura will have become thickened to 1.5 cm. or more. On the other hand, the fibrous tissue membrane covering the lung in these cases is never more than 3 mm. in thickness; consequently, great care must be exercised in the original incision of the membrane.

I have made it a practice to incise longitudinally, that is to say from top to bottom of the exposed surface, using practically no pressure other than that of the weight of the scalpel. Depending upon the width of the surface which requires decortication, two, three, or even four longitudinal incisions of this sort are made. Again depending upon the length of the exposed surface, similar incisions are carried across, cutting the original incision at right angles.

Such a method of preliminary incision of the membrane presents the operator with a number of right-angled points. These are checked over so that the angle at which the parietal pleura is most clearly identified can be used as a guide in determining the exact thickness of the membrane.

By means of blunt dissection, with, from time to time, the employment of a scalpel, it is usually not very difficult to remove the membrane in patches such as have been marked out. In my experience, it is almost impossible to decorticate in this way the whole exposed lung surface without damaging the lung itself at various points. Bubbles of air, therefore, are likely to appear and, from time to time, slight bleeding. The bleeding which occurs is very rarely in the slightest degree alarming and can be confidently expected to cease with hot saline gauze packing for a few minutes. It would seem to be true that such superficial damage to lung tissue heals rapidly.

Once the dense, fibrous, tissue membrane has been removed from the surface of the lung to the edge of the superficial portion of the latter, it is then necessary to continue the dissection between the lung and the parietal surface. This separation is comparable to that of dissecting free the lung in performing lobectomy for bronchiectasis, and is consequently carried out by means of scissor dissection, both blunt and sharp. This dissection should be carried as close to the hilar region as possible if adequate expansion of the lung is to be obtained.

Although it is true that interstitial fibrosis of the pulmonary tissues may occasionally occur, so that expansion of the lung will not take place, this must, I believe, be unusual.

In the case of chronic empyema consequent upon tuberculosis of the upper part of the upper lobe, it would seem to be a matter of wisdom to avoid extending the decortication into the area in which massive tubercle formation is to be expected. It is suggested, therefore, that in such cases collapse of the apical portion of the chest be obtained by means of a classical extrapleural thoracoplasty.

If the operation has been carried out under local anesthesia, it is a simple matter to induce the patient to cough; if, however, an inhalation anesthetic has been employed, it is necessary and wise to permit the patient to regain consciousness. Almost invariably the lung will be seen to expand under the increased pressure of coughing, and, since the necessary rib resection which has preceded the operation of decortication and the contraction of the chest in consequence of the fibrothorax has diminished the capacity of the diseased half of the thorax, as a rule the lung will not only fill the chest cavity but attempt to herniate through the opening.

It is essential, I believe, that the wound of the interior of the chest be absolutely free from oozing before the completion of the operation. Rarely are ligatures required for this purpose, since packing with hot saline gauze can be expected to arrest practically all bleeding.

Before closing the chest we have made it a practice to provide for "curtain drainage" over the whole surface of the lung, which, as indicated, is almost certainly the site of superficial damage at varying points. For this purpose, two or three thicknesses of soft dressing gauze is soaked in liquid paraffin to which a small amount of "bipp" has been added, or sulfathiazol emulsion prepared according to our formula[5] is generously applied to the surface of the gauze. This apronlike covering of gauze is brought out through the lower corner of the wound, which is otherwise closed by means of through-and-through silkworm gut sutures.

The gauze is removed at the end of the sixth or eighth day. Every attempt is made to induce the patient to cough vigorously at short intervals throughout the day. The patient is, moreover, urged to get out of bed as soon as possible and to exercise to the utmost.

Following the removal of the gauze, the wound is permitted to heal and the chest is very carefully watched so as to be certain that fluid does not reaccumulate in any considerable quantity. In the third of the three cases which constitute the basis for this contribution, relative failure is believed to be due to oversight in this connection.

There has recently been published in Paris, by Masson et Cie., a six-volume treatise on surgical technique, of which the third volume deals with surgery of the thorax. This volume has been written by Dr. Marc Iselin, who is personally known to many members of this Association, and who read before the Association at the Washington meeting in 1934 a paper on pulmonary abscess prepared by Professor Sergent and himself. This volume of Iselin's constitutes the most up-to-date publication on thoracic surgery, and, since Iselin is familiar with the literature published in English as well as French, it constitutes a valuable contribution.

The following is the author's translation of a section devoted by Iselin to the subject of decortication. "Pulmonary decortication, also known as the operation of Delorme, has as its aim the removal of the covering which holds the lung over the whole of its surface. Once it has been carried out, according to the expression used by its author, it permits the lung to herniate *comme un ballon brusquemont gonflé.*

"Having satisfactorily exposed the visceral covering at the bottom of the pleural cavity, well spread by an automatic rib spreader, the middle of this surface is incised; cutting very carefully millimeter by millimeter with the scalpel, which cuts more regularly than the cutting current. After having traversed the waxlike covering of the thickened pleura, the lung appears easily recognized; it then becomes necessary to find the line of cleavage which never appears to be absent between the lung and the visceral covering, using a spatula or a soft instrument. If this line of cleavage is well established, the decortication is easy and not complicated by bleeding. Each of the four leaves decided upon is raised with the help of traction (quadrants) and with a spatula blunt dissector one uncovers in the depths; the lung is then seen to expand. If the operation is carried out under favorable conditions, it is possible thus to go to the angles and to liberate them around the periphery. It is clear that this is the type of case in which the *gonflement brusque* described by Delorme is obtained.

"Unfortunately, this technique in many cases seems to be very theoretical. It is probable that usually it is not possible to find the cleavage line, because in the descriptions, and in the figures published, the authors recommend not to uncover too much, to tamponade the damaged surface of the lung when it bleeds, and to know when, in the individual case, not to carry on. Without question, for these reasons Duverger has also recommended not to attempt to raise the thickened pleura but to be content to incise *en herse* by a series of scarifications, going just to the lung, in a manner in which the quarters determined meet with one another and permit the expansile tendency of the lung to show itself.

"I must state that all this seems very illusive to me; I once saw a pleural decortication a la Delorme by a foreign surgeon who had always been an ardent defender of the procedure; but despite great care, and despite attention to the minutiae of his operation, after a few minutes he had made a large opening in the diaphragm (an opening which was, nevertheless, immediately sutured) and later carrying out the decortication, he opened the pericardium; at this time he stopped the operation, which up to this moment had not seemed to me very valuable."

"I have examined a certain number of pleural fistulas after extensive thoracotomy but I have never felt the least temptation to carry on and incise the visceral pleura in order to commence a decortication. Also, I have not been able to know the limits of the lung or where the diaphragm or the mediastinal structures commence. Finally, there is the last argument against decortications: in peacetime surgery one should not forget that one of the great causes of pleural fistulas, even after empyema of an apparently harmless variety, is underlying tuberculosis; there is the risk, therefore, of decorticating a tuberculous focus, with all the dangers that this represents."

This ends Iselin's comment regarding decortication in chronic cases. It is evident, as he himself states, that he has had no experience in performing the operation. I do not wish to suggest that the operation is always easy, that the results are perfect, or that there is no risk of damage to the victim, on the other hand, I believe that opinions such as those expressed by Iselin and others have interfered with an extended use of the procedure and that, in consequence, its proper evaluation has been deferred.

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DISCUSSION

DR. DANIEL A. MULVIHILL, New York, N. Y.—The recently aroused interest in the old operation of lung decortication, brought about by the wartime experience with fibrosing hemothorax, has naturally turned the thoughts of many of us to its possible applicability in the much wider field of pulmonary tuberculosis.

Dr. Gurd's cases today have shown us that it is not only desirable but feasible in certain types of cases to get a re-expansion of the lung in tuberculosis.

The case that I am to present illustrates the application of decortication to another tuberculosis problem, that of the nonexpanding lung following long-standing pneumothorax, when a new cavitary lesion on the opposite side demands surgical collapse therapy.

(Slide.) This x-ray represents the problem presented by a 28-year-old married white woman on her readmission to Triboro Hospital in September, 1945, with cavitation in the right upper lobe. Her original onset of symptoms dated from April, 1944, followed by hospital admission in June, 1944, pregnant and with a large, thin-walled cavity in the left upper lobe.

Pneumothorax was induced early in July, 1944, and was satisfactory as regards sputum conversion, but there were some adhesions, part of which were divided by pneumonolysis in September, 1944. Normal delivery took place in October, 1944. In February, 1945, a second pneumonolysis resulted in a complete collapse of the upper lobe, and the patient was discharged from the hospital in April, 1945, continuing pneumothorax refills as an out-patient.

Following readmission in September, 1945, because of the cavity in the right upper lung, refills on the left side were stopped, and attempts were made to induce pneumothorax on the right. These were unsuccessful.

In a period of six months of bed rest the left lung failed to expand and presented the picture shown here when the patient was brought before the surgical conference (during the latter part of March, 1946) for thoracoplasty for the right upper lobe lesion.

The patient was dyspneic on very slight exertion. Her vital capacity was 1,400 c.c., and bronchospirometric studies showed that the left side was providing only 15 per cent of the total functional capacity. Obviously, she was not a suitable risk for the amount of rib resection necessary to collapse the cavity in the right upper lobe.

Decision was made to attempt decortication and re-expansion of the left lung. A preliminary thoracoscopy was done and the pneumothorax space was seen to be lined with a pearly white, fibrous envelope. No lung or thoracic landmarks could be distinguished. Thoracotomy was then performed with resection of the posterior portion of the fifth rib, and the pneumothorax space was entered through its periosteal bed. The space was dry, but the pearly surface was slightly moist and firm, grossly not unlike that found in fibrosing hemothorax. Incision was made through this fibrous membrane, which was about one-eighth inch in thickness, down to the visceral pleura. A line of cleavage was found between the intact and normal appearing visceral pleura and the inner surface of the fibrous plaque. This line of cleavage was composed of loose, areolar, slightly vascular adhesions, which could be easily separated with gauze dissection or the finger. Near the apex, an area about two inches in diameter was encountered where the plaque became intimately adherent to the underlying lung. Slight discoloration and a pebbly appearance of the surface in this area indicated that there was tuberculous pathology immediately beneath, which would probably be entered if dissection were attempted. Separation of the plaque from the lung was carried out all around this area as far as the adhesions were loose and easily separated. Then the plaque was cut around it, and this fibrosed area was left in place on the surface of the upper lobe. The fibrous plaque was then divided all around the periphery at the angle of its reflection onto the parietal pleura. It was not deemed necessary to remove the parietal element. The lung was then entirely freed from the mediastinum and the diaphragm, and the fissure was separated. The lung was expanded by intratracheal positive pressure and it filled the entire chest cavity. No. 40 French right-angled drainage tubes were then placed through the third anterior and seventh posterior interspaces, and connected with waterseal bottles. Thoracotomy wound was closed in layers with black silk sutures. The lung fully expanded and tubes were removed in forty-eight hours.

Her postoperative course was uneventful.

(Slide.) This tomograph was taken the day before operation, showing the extent of expansion of the left lung that could be obtained after six months of bed rest.

(Slide.) This is the fibrous plaque removed at operation. This surface is the surface facing the pneumothorax cavity.

(Slide.) This is the surface that adhered to the lung, showing the loose layers in which the dissection can be carried out. The notch on this side indicates where the plaque was so adherent to the lung that it was cut around and left in place.

(Slide.) This shows the microscopic appearance showing the fibrous nature of the plaque. You will note that there is no evidence of infection and that there are no tubercles visualized. That was true in many other slides made from this material.

(Slide.) This x-ray shows the re-expansion obtained postoperatively. As I said, the patient's postoperative course was uneventful. The operation was performed about six weeks ago. She is now able to walk and exercise without dyspnea, her vital capacity has increased from 1,400 c.c. to 1,900 c.c. and we now feel we can go ahead with thoracoplasty on the right side.

I would like to emphasize that the limitations of this operation, whether it is done for tuberculosis or any other reason, probably depend on whether the pathology is in the visceral pleura itself, or, as in this case, is a fibrotic sheet superimposed on an essentially normal and intact visceral pleura.

DR. E. J. O'BRIEN, Detroit, Mich.—Two months ago Dr. Gurd and I were in Boston, and I was very enthusiastic about the work of the young men who were just returning with news of what I thought was one of the best contributions that had come from the war, decortication. Many years ago, those of us interested in thoracic surgery who tried it quite extensively on old chronic empyemas had mostly given it up. Dr. Gurd said that he had been doing it all these years and had always obtained very good results. Therefore, I asked him to show us the results which he had obtained; he has limited his paper to tuberculous empyema, so I have not been convinced.

With the new and recent stimulus, we have tried again quite extensively to see what its limitations were. The procedure has been quite effectual in most of the early tuberculous and other empyemas. It is a great help in upper lobe cavities that have been held down with pneumothorax for a long time when we wish to discontinue the pneumothorax. One can frequently decorticate and re-expand the lower lobe and have the upper one stay down. This is especially desired if there has been an extensive disease in the upper lobe. Decortication of lungs to re-expand them when extensive tuberculous disease has been present is a hazardous procedure. We have again tried it in the much older and more chronic empyemas, where my previous experience has been disappointing, and we are running into the difficulties I ran into many years ago. We are getting a lot of bubbles from the lung and it often does not re-expand. I have had one patient who died.

As Dr. Gurd said, you can tear into the lung, and I did.

I think you can expect results in direct proportion to how far the infection and cicatricial tissue has invaded the interstitial tissue.

In the early cases, such as have been reported from the war, when the so-called pleura is removed, you have a nice shiny, glistening pleura, but in the old chronic cases that is not the case as a rule.

I am in favor, of course, of trying decortication on all of these patients, and I hope we are going to get better results than we have in some of those we have attempted.

I wouldn't say that in tuberculous empyema six months should be called very chronic, because there are so many patients that have been badly treated with pneumothorax and have gone for years with aspiration. Many of these patients should not be decorticated because of previously existing extensive tuberculous disease. I would like to think that we can get lungs re-expanded in very old chronic empyemas in a good percentage of patients, but I am dubious.

When the doctors came home and showed us all the work they had done, I thought I had been making a mistake all these years, but I am not yet convinced. We are going to try it faithfully but there must be limitations.

I want to warn you that we already are running into some trouble in old chronic empyemas. I believe they will not turn out as did the earlier cases that were shown by the boys who came back.

DR. CARL EGGERS, New York, N. Y.—Dr. Gurd stated that the first cases of tuberculous empyema which he treated by means of decortication were among the residue of chronic empyema patients resulting from the first world war.

We, here in the U. S. A., had a similar experience. A large series of chronic empyema patients were subjected to critical analysis in order to find the reason for nonhealing, whether it be imperfect drainage, secondary pockets, bronchial fistula, foreign body, or some other cause. Among these other causes, unrecognized tuberculosis, either apparently limited to the pleura, or associated with parenchymal disease on the same or opposite side, was occasionally found to be the responsible factor.

In the *Annals of Surgery* for March, 1923, pages 327 to 353 and 375 to 378, we reported the results obtained in 146 patients with chronic empyema treated radically by means of decortication of the lung. Among these were eleven patients in which positive evidence of tuberculosis was obtained after radical operation. Of these eleven, three healed, one healed and died one year later, three did not heal and later died, and four were lost from follow-up.

The impression we gained from these cases prompted us to apply the same procedure in six cases of chronic empyema which we were able to prove to be of tuberculous origin, prior to the radical operation. Three of these healed and were apparently also cured of their intrapulmonary focus, two continued to drain, and one was lost from follow-up.

The following is a typical illustrative case: A woman of 24 was admitted with chronic empyema of nine months' duration, the result of an intrapulmonary complication following childbirth. Our examination disclosed the following facts: There was no temperature or increased pulse, no cough or expectoration; discharge showed streptococci and tubercle bacilli, skin reaction was positive for tuberculosis, biopsy of sinus showed tuberculosis, a tuberculous focus was noted in the apex of the opposite lung, and injection of the cavity showed it to extend from the ninth to the fifth rib. After preliminary Dakin-Carrel treatment, a typical radical procedure was carried out in two stages. The object was to mobilize the chest wall, to mobilize the lung, and to remove all diseased tissue. To do this, small pieces of seven ribs were resected and the parietal pleura and the angle of reflection onto the lung removed in order to mobilize the lung. An incomplete decortication was done for fear of entering an intrapulmonary focus, and no attempt was made to inflate the lung. Convalescence was uneventful. The wound healed and the patient is known to have been entirely well twenty years later.

An interesting point connected with this case is that the boy who was born when the mother contracted her disease developed tuberculosis of the spine for which a fusion was done with a perfect result.

It is my feeling that although we speak of and perform a radical operation with decortication in these cases, we should compromise somewhat. Rather than do a complete decortication and forcible expansion of the lung, which may cause lighting up of the disease or perforation of a subpleural tuberculous focus, it is better to be satisfied with mobilization of the chest wall and lung as well as a thorough removal of all diseased tissue.

I have been sufficiently impressed with the value of the operation in these cases to continue to advise it in the occasional patient that comes under observation.

When it comes to applying the principle of decortication to typical pulmonary tuberculosis associated with a draining empyema of tuberculous or mixed infection, I would like to sound a note of warning. The prognosis depends chiefly on the extent and severity of the underlying intrapulmonary disease, and is frequently hopeless. Only if it is felt that the parenchymal disease is under control and the draining empyema is the chief lesion, operation is indicated, but should not consist of decortication, but of mobilization of the chest wall by the resection of short pieces of ribs overlying the cavity and complete unroofing of the cavity with excision of the angle of reflection onto the lung. One has to depend here not on reexpansion of the diseased lung, but on sterilization and mobilization of the chest wall in order to bring about healing.

There is a third group of patients which deserve consideration, namely, patients with tuberculosis of the upper lobe who have been on prolonged pneumothorax treatment, but with an unsatisfactory result. Surgeons are frequently called upon to see such patients. The lower uninvolved lobe is beautifully collapsed, has become fixed in that position, and is permanently out of function, while the upper diseased lobe has not collapsed because of fixation to the chest wall. Evidently the pneumothorax treatment has been continued beyond the time that seems justified. An extrapleural thorocoplasty will take care of the disease in the upper lobe, but what to do with the lower lobe which should never have been collapsed, or rather, which should not have been allowed to remain collapsed so long, is a problem. Sometimes gradual re-expansion will take place, but if it remains collapsed it constitutes a problem for the younger men of our association to solve. Perhaps a decortication limited to the lower lobe will prove feasible and practical, and not associated with too much risk. To apply the general principle of decortication to pulmonary tuberculosis without careful consideration and the cooperation of internists would be a mistake.

DR. RICHARD H. MEADE, JR., Chicago, Ill.—It is extremely important that this subject has been brought up for discussion. Dr. Eggers has covered all of the points I was going to ask that he cover, except for one small technical point.

When Dr. Eggers presented his paper in 1920, which later was published, I read it with a great deal of interest, and some years after that had occasion to apply his procedure in a patient that I thought had a nontuberculous empyema. Everything went beautifully and the patient recovered quickly, but unfortunately returned with widespread tuberculosis and died.

The next patient with unrecognized tuberculous empyema on whom I did a decortication (during the war) also died with widespread tuberculosis.

I think that if one can be sure in which cases the decortication can be done without that great danger, it can be applied quite widely.

There is only one very minor technical point that I think is worth mentioning, because it has been brought up a number of times, and that is, everyone has spoken of the necessity of resecting a number of ribs in these cases in order to get adequate exposure. If one will simply strip off the greatly thickened layer over the parietal pleura when the first incision is made, over a space of several ribs, one can get adequate exposure without resecting large segments.

I believe that the use of adequate drainage, as has been shown frequently in chronic empyemas of nontuberculous nature, and also in the chronic pneumothorax cases, is of great help in bringing about complete and rapid reexpansion.

I believe, however, the most important question is, in which cases of tuberculous empyema can one carry out this procedure without the danger of stimulating spread of the disease.

DR. FRASER B. GURD, Montreal, Que.—It was naturally impossible in the time at my disposal for me to go into the pathology of tuberculosis or into the life-history of the tuberculous. In my original correspondence with the Secretary, I pointed out that there were but three cases but that, if it were deemed advisable by the Programme Committee to have this subject presented for discussion, I should be glad to do so.

I should like to thank those who have taken part in the discussion.

CAVERNOSTOMY

E. J. O'Brien, M.D., P. V. O'Rourke, M.D., and (by invitation)
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ADEQUATE treatment for the greater number of patients with pulmonary tuberculosis can be provided by bed rest in combination with a few well-standardized procedures such as pneumothorax and supplemental pneumonolysis, phrenic surgery, and thoracoplasty. A considerable group of patients remains, nevertheless, for whom standard collapse therapy is either unsuccessful or inapplicable.

The literature of the past two decades contains many references to the management of cavernous tuberculosis in patients with giant cavity or tension cavity; with bilateral cavities or lower lobe cavity; with residual cavity after collapse therapy; or with dyspnea or advanced age as a barrier to routine management. Various new methods have been suggested to deal with some of these circumstances. Along with procedures such as extrapleural pneumothorax, paraffin plombage, apicolysis of Semb, Monaldi endocavitary aspiration, and, most recently, pulmonary resection, surgeons are finding a place for open surgical drainage of the tuberculous cavity, or cavernostomy.

The present report is based on eighty-four cavernostomy operations* performed at the Herman Kiefer Hospital, and at three near-by tuberculosis sanatoriums. Seventy-four patients were operated upon. One had bilateral operations and several others had secondary drainage at the same site. Up to the present time, we have used cavernostomy chiefly as a salvage measure when other procedures were contraindicated or had failed. For a few patients, it was used electively.

HISTORICAL BACKGROUND

The surgical drainage of tuberculous pulmonary cavities is not new. It is probably very old. In an interesting historical survey of the procedure, Vineberg and Kunstler[22] suggest that Hippocrates may have described this procedure, though his writings perhaps refer to the open drainage of an *empyema necessitatis*. These authors also point out that Baglivi[2] in the late seventeenth century held the opinion that the opening of cavities leads to cicatrization. The undeveloped state of pathologic anatomy at that time suggests that the lesions treated may have been pulmonary abscesses or loculated empyemas.

In the eighteenth century a Dr. Barry[3] of Dublin wrote of the beneficial effects from surgical puncture of tuberculous cavities in several patients. Reports of cases at this time are more likely to be authentic, since both surgical knowledge and recognition of morbid anatomy were more advanced. The

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*A number of the operations in this series were done by W. M. Tuttle, M.D., and J. C. Day, M.D., before their entry into the armed forces.

English surgeon Sharpe,[10] writing at nearly the same period, undoubtedly referred to drainage of intrapulmonary lesions and knew the importance of pleural adhesions in reducing the difficulties which followed surgical drainage across a free pleural space.

According to Elliott,[8] Sir Henry Marsh of Dublin may have drained tuberculous cavities in two patients upon whom he operated about 1830. Recovery followed in one of these. Hastings[8] with the help of a surgeon, Mr. Stokes, opened one tuberculous cavity in 1844 and later performed a second similar operation. The wounds, first packed with lint or gauze, were later drained by a rubber tube. Hastings reported considerable improvement in these two instances and also after a third similar operation by Mr. Thomas, a surgeon of Wales. He pointed out, however, that the procedure should be limited to the occasional patient with a large, solitary cavity and a relatively sound contralateral lung.

In 1885, De Cerenville expressed the belief, according to Vineberg and Kunstler,[22] that cavity drainage would prove to be a useful operation. These authors also cite Sauerbruch and other continental surgeons as holding a similar opinion. The rapid development of surgical technique and surgical experience in the latter years of the nineteenth century soon made it obvious that drainage of tuberculous abscesses in any part of the body left chronically draining wounds and yielded disappointing results. Pulmonary cavities have been opened deliberately or inadvertently numerous times in the early period of modern thoracic surgery. The attendant disadvantages and complications made avoidance of the procedure almost axiomatic, and the development of collapse therapy demonstrated the relative superiority of extrapulmonary thoracic operations over intrapulmonary drainage.

In the early twentieth century, cavity drainage was so little used that Gekler and his associates,[11] who, in 1924, tried cavernostomy tentatively on three very ill patients, noted with surprise the favorable effect and attributed it largely to the "sterilizing" influence of coal-tar dyes which they used by local application. They reported progressive reduction in the size of the cavity and early appearance of granulation tissue in the cavity and surgical wound, with general improvement in each patient.

Lilienthal,[13] in 1927, reported open drainage of a large blocked cavity remaining after thoracoplasty. The patient's condition improved, the cavity became indefinite, and the wound presumably healed. Two years later this author stated[14] that the patient was further improved though the wound reopened. He also cited two additional cases of post-thoracoplasty cavity subjected to open drainage. Sputum examinations are not reported in one, but the wound almost closed, leaving a small sinus. In the other, the cavity was opened into a residual empyema and sputum examinations became negative while healing progressed considerably with only two small fistulas remaining.

Nissen,[16] in 1931, gave a short report of one patient on whom cavernostomy was followed by transplantation of a pedicled graft into the cavity. The cavity appeared closed and the postoperative photograph shows firm healing of the wound.

A description of the flap-valve mechanism of blocked cavity was given in a study of pathologic specimens by Hall[12] as early as 1922, but only in the decade just past have we reached a real understanding of the effect of the efferent bronchus in the closure of cavities by collapse therapy. The work of Coryllos[5] in 1936 on the mechanics of tuberculous cavities was outstanding. He came to believe all rapid changes in the size of cavities and their healing or resistance to closure, were due to the state of the bronchial outlets. Coryllos and Ornstein[6] made detailed studies of cavities in the living by endoscopic examination, and concluded that healing would follow complete bronchial occlusion, which they attempted to produce.

Eloesser[9] and others likewise recognized the role of a valvular bronchus in tension cavities, but disagreed as to the value of closing the bronchial outlet. The point was generally established, however, that a relation exists between the state of the efferent bronchus and the response of a cavity to collapse therapy. A rational basis was thus provided for an intrapulmonary attack on certain cavities which resisted closure by collapse therapy.

In 1937, Eloesser[9] reported the performance of cavernostomy for tension cavity in two patients. Open drainage in his first patient was done after thoracoplasty. At the time of his report there was clinical improvement but the cavity was open and sputum was positive. In the next instance cavernostomy was used for bronchopleural fistula after paraffin plombage. The wound and cavity were opened and packed with gauze, and later healed although the patient retained a productive cough. Postoperative sputum examinations are not mentioned.

Coryllos and Ornstein[6] drained twelve cavities which remained open after thoracoplasty, and performed early closure with a pedicled muscle graft. The sputum became negative for tubercle bacilli in five, one of whom died later of pneumonia. Three other patients also died. Their report makes no mention of the postoperative x-ray appearance and ultimate cavity closure in the series.

Neuhof[15] used free grafts of fat to close pulmonary cavities in a group of patients of whom only two were tuberculous. In one of the latter, a cavity persisted after thoracoplasty, and was opened and packed with loose fragments of fat. The wound healed satisfactorily and sputum examinations became negative. In the second tuberculous patient, the grafts were infected and expelled. The final result is not mentioned.

Brunn and his associates,[4] in 1941, reported one open drainage of a residual cavity after thoracoplasty. A skin flap was sutured to the pleura and the wound later healed. The result was considered successful. Eloesser,[10] about the same time, described two cases of cavity which failed to respond to the Monaldi procedure of cavity aspiration. Open drainage was done with the creation of a skin flap, and the wounds healed, leaving a bronchial fistula. He does not mention sputum conversion nor final complete healing.

Steele[21] mentioned one patient who had drainage of a cavity not closed by an eight-rib thoracoplasty. At the time of his report, the cavity and wound appeared healed but sputum examinations were not mentioned. Davis[7] reported that his patient, after a similar procedure, had negative sputum with a partly healed wound four months after operation.

Rogers, Shipman, and Daniels[18] performed flap drainage of a cavity on nine patients after thoracoplasty, with no deaths. Five of these had healed wounds and negative sputum, and another had negative sputum less than six months after operation. Randolph[17] has recently reported cavernostomies on two patients after unsuccessful attempts at extrapleural pneumothorax. In one the sputum became negative and the cavity appeared closed. The state of wound healing is not mentioned. In the other the cavity appeared to close following open drainage. No mention is made of sputum examinations or wound healing.

The reports of these surgeons, and other passing references such as that of Alexander,[1] indicate that open surgical drainage of cavities is being done more frequently now than in former years. The indications for cavernostomy, however, are not yet well defined. The discussion of our cases may show what can be expected of the operation in the field of tuberculosis surgery.

INDICATIONS FOR CAVERNOSTOMY

The patients we selected for this procedure were drawn mainly from three well-defined groups; a few miscellaneous patients make up a fourth group. The classification is shown in Table I. The most common indication for cavernostomy was an inadequate cardiorespiratory reserve which precluded other forms of surgery. This condition was present in the forty-three patients of Group I. In twenty-six, slight or moderate dyspnea was present at bed rest. In the other seventeen individuals, respiratory deficiency was established as present by tests of exercise, by differential bronchospirometry, or by previous attempts at collapse therapy. All but one of these forty-three patients had preexisting tuberculosis in the contralateral lung, and thirty-one (72 per cent) had previous contralateral collapse therapy, with resulting impairment of pulmonary function. Such measures are listed in Table II and include, in the order of relative frequency, unexpanded or re-expanded pneumothorax with thickened pleura and encased lung; permanent or residual phrenic paralysis; uncomplicated pneumothorax; and thoracoplasty. Of the other twelve patients (28 per cent) eleven had extensive contralateral productive lesions, fibrosis, or emphysema, and each was slightly dyspneic. The last patient appeared to have one lung clear, but had auricular fibrillation and was dyspneic at bed rest.

Three-fourths (74 per cent) of those in Group I also had previous ineffective attempts at collapse therapy on the side of cavernostomy, as shown in Table III. Twenty-nine (67 per cent) received an initial trial of pneumothorax; fourteen of these were given pneumothorax alone; ten, a combination

TABLE I

GROUP	INDICATION FOR CAVERNOSTOMY	NO. PATIENTS	PER CENT
I	Inadequate cardiorespiratory reserve		
	A. Noticeable dyspnea	26	35
	B. No dyspnea but impairment of contralateral lung	17	23
		—	—
	Total	43	58
II	Residual cavity after thoracoplasty	13	18
III	Lower lobe cavity	8	11
IV	Other conditions	10	13
	Total, all cases	74	100

TABLE II. COLLAPSE THERAPY OF CONTRALATERAL LUNG PRIOR TO CAVERNOSTOMY (PATIENTS WITH LOW CARDIORESPIRATORY RESERVE)

PROCEDURE	NO. PATIENTS
Pneumothorax, with thick pleura and encased lung	5
Pneumothorax, re-expanded, with thick pleura and encased lung	9
Pneumothorax, uncomplicated	6
Phrenic paralysis, permanent or persistent, after crushing of nerve	9
Thoracoplasty	2
None (extensive pulmonary emphysema or fibrosis)	11
None (lung clear but patient dyspneic)	1
Total	43

of pneumothorax and phrenic paralysis; and five, phrenic surgery alone after initial pneumothorax failed to demonstrate a free pleural space. Three additional patients (7 per cent) had a partial, inadequate thoracoplasty interrupted because of dyspnea. Two (4 per cent) had previous pneumoperitoneum. The remaining nine (22 per cent) had no previous attempt at pneumothorax on the side of cavernostomy either because of dyspnea or because of the large size of the cavity. In our experience, pneumothorax collapse for giant cavity has been followed by serious complications in from one-half to three-fourths of the patients.

TABLE III. COLLAPSE THERAPY OF IPSILATERAL LUNG PRIOR TO CAVERNOSTOMY (PATIENTS WITH LOW CARDIORESPIRATORY RESERVE)

PROCEDURE	NO. PATIENTS
Pneumothorax, re-expanded	14
Phrenic paralysis after unsuccessful pneumothorax	5
Pneumothorax plus phrenic paralysis	10
Partial thoracoplasty (incomplete because of dyspnea)	3
Pneumoperitoneum	2
No collapse therapy	9
Total	43

These members of Group I, then, had visible or latent dyspnea. They had rather long-standing bilateral tuberculosis, and ample but unsuccessful previous treatment. No additional measures of collapse therapy seemed applicable, and the prognosis was uniformly poor.

Group II comprises thirteen patients with residual cavity beneath a thoracoplasty (Table I). Nine of these (69 per cent) had had one or more revision thoracoplasty operations and were thought to have maximum surgical collapse. Three (23 per cent) showed globular cavities up to 8 cm. in size after a three-stage thoracoplasty with removal of seven ribs. One (18 per cent) had a large tension cavity beneath a partial thoracoplasty, after removal of five ribs. None of these patients had dyspnea at rest. Three had definite exertional dyspnea, but had undergone the previous surgery without difficulty.

Nine members of this group could be considered as affording a tolerable risk for any major surgery. The three with exertional dyspnea, and one other with active exudative lesions about the tension cavity, appeared to offer a poor risk for drastic surgical intervention.

Eight patients in Group III each had a large cavity in a lower lobe, with adequate respiratory reserve and a clear or nearly clear contralateral lung (Table I). All of these patients had a previous trial of pneumothorax or phrenic paralysis or both. One young diabetic male developed the lower lobe cavity three years after a seven-rib thoracoplasty which had closed an apical cavity on the same side. One man aged 67 years, was thought to be a poor risk for more radical surgery. The other seven were in satisfactory general condition for any surgical procedure.

In two of the ten miscellaneous patients in Group IV (Table I), the residual bronchocutaneous sinus after a Monaldi operation was converted to open cavernostomy for the control of persistent bleeding from the chest wall and cavity. Both of these men also had active tuberculosis and cavities in the opposite lung. In two patients with huge apical cavity, Monaldi intercostal drainage was done after an anterior thoracoplasty and was followed by posterior thoracoplasty without cavity closure. The residual cavity and sinus were converted to open drainage. Cavernostomy was used in one instance of large bilateral apical cavities with the expectation of later performing thoracoplasty on the other side. For three elderly men with huge cavity and intractable cough, cavernostomy was done as a palliative measure. One young man had an unhealed posterior wound and bronchocutaneous fistula, following an infected three-rib thoracoplasty, and an infected anterior chest wall adjacent to a Monaldi catheter drainage (both performed elsewhere), with a large residual cavity. This was subjected to open drainage to divert the stream of infective material from the previous wounds. One child of eight years with dense exudative tuberculosis of one lung surrounding a 4 cm. lower lobe cavity, and with ulcerative tuberculous enteritis, was chosen for cavernostomy after pneumothorax was attempted without success.

The members of Group IV all had active scattered or dense exudative lesions in one or both lungs at the time of operation. It seemed clear that these patients, like those in Group I, had an apparently hopeless prognosis under the usual methods of management.

The seventy-four patients discussed here all had advanced tuberculosis. In five the disease was moderately advanced; in sixty-nine, far advanced. Only five had bilateral open cavities at the time of the cavernostomy operation. None had more than one cavity in the lung operated upon (though one later developed a second cavity in the same lung). Of the total number, sixteen (21 per cent) were in favorable condition for major surgery, although all of them had undergone an adequate trial of collapse therapy without benefit. The remaining fifty-eight (79 per cent) are shown to have been unfit for any surgery which might decrease the cardiorespiratory reserve. They had been given an unfavorable prognosis.

SURGICAL TECHNIQUE

Anesthesia.—The anesthesia used was either local infiltration or general anesthesia by nitrous oxide and oxygen. The choice between these was dependent upon the condition of the patient and the preference of the individual surgeon.

TABLE IV. OPERATIVE PROCEDURES

PROCEDURE	REASON	NO. OPERATIONS		PER CENT
Single stage	No free pleural space		44	53
Multiple stages	Free pleural space over cavity	17		
	Inadequate localization of cavity	8		
	Dense plate of bone over cavity	1		
	Preliminary packing of wound to avoid infection of chest wall	1		
		—		
	Total		27	32
Secondary drainage	Wound partly healed. Cavity open	1		
	Cavity reopened under healed wound	4		
	Cavity reopened after plastic closure of wound	2		
		—		
	Total		7	8
Incomplete operation	First stage followed by pleural effusion and cavity closure	2		
	Small cavity not located	2		
	Secondary drainage attempted. Small residual cavity not located	1		
	Fatal air embolus after first stage operation	1		
		—		
	Total		6	7
Total			84	100

Location of Incision.—An attempt was always made to approach the cavity at the point nearest the chest wall. In the eighty-four operations, a lateral approach was used six times; an anterior approach, ten times; a posterior, sixty-eight times. The incision was straight or slightly curved. Creation of a plastic skin flap of the Eloesser type was not done. The location of the cavity in these operations was as follows: right upper lobe, 38 per cent; left upper lobe, 32 per cent; total, upper lobes, 70 per cent; right lower lobe, 21 per cent; left lower lobe, 9 per cent; total, lower lobes, 30 per cent.

Rib Removal.—The length and number of ribs removed was usually merely enough to unroof the area of the subjacent cavity. This was most often a 4 to 6 cm. length of one or two ribs. In several operations for lower lobe cavity on "good-risk" patients, longer lengths of three ribs were deliberately removed to allow more relaxation of the area of pulmonary disease.

Opening of Cavity.—When a partially free pleural space was present, the wound was packed with gauze for a number of days before the pleura and cavity were opened at a second, or occasionally a third, stage. An electrocautery blade was used for incision of the lung. A few of the opened cavities were packed with gauze throughout the postoperative period. In the rest, a firm rubber tube was introduced and maintained until the bronchial fistulas appeared closed.

Other Surgical Procedures.—In eight instances (seven patients), firm healing of the chest wall or the skin occurred before the cavity closed, or the closed cavity reappeared some time after healing of the wound. Secondary cavernostomy operations were then performed. In five patients with small (2 cm.) cavities, the operations were incomplete and the cavity was never successfully opened. On two occasions we closed the granulating wound and cavity by a plastic operation with a pedicled muscle graft.

Of the eighty-four operations, forty-four (52 per cent) were performed in one stage, twenty-two (26 per cent) in two stages, and five (6 per cent) in three stages. There were eight (10 per cent) secondary revision operations (one incomplete). Five primary operations (6 per cent) were unsuccessful without opening into the cavity. These procedures are listed and explained in Table IV.

The type of procedure was determined by various circumstances. Obliteration of the pleural space at the operative site was encountered forty-four times and permitted immediate opening of the cavity. A two-stage or three-stage operation was required seventeen times because of a free pleural space. In eight operations, inadequate localization of the cavity

TABLE V. DISTRIBUTION OF CAVERNOSTOMY OPERATIONS

PROCEDURES	NO. PATIENTS	NO. OPERATIONS
Single cavernostomy (one or more stages)	60	60
Bilateral cavernostomy	1	2
Two cavernostomies for separate cavities in same lung	1	2
Primary cavernostomy and secondary drainage of cavity	5	10
Primary cavernostomy and two secondary operations	1	3
Incomplete operation*	6	7
Total	74	84

*One patient had had a previous cavernostomy with premature wound healing.

required multiple stages; after further x-ray studies, the cavity was successfully drained. In one instance, a dense plate of bone following a previous thoracoplasty required a two-stage operation to drain the underlying cavity. Preliminary wound packing with second-stage cavity drainage was done once after wide rib resection over a large lower lobe cavity to avoid a spreading infection of soft tissues.

A secondary or revision cavernostomy was done once to enlarge an inadequate stoma to a large cavity. Two healed wounds following plastic closure, and five which healed spontaneously, were reopened to drain the recurrent cavity. In one of the latter operations, we did not succeed in reopening the small residual cavity and this operation is listed as incomplete. Of the five other incomplete operations, two were interrupted after the first stage by a traumatic pleural effusion in the partially obliterated pleural space. In these patients the cavity then disappeared on x-ray examination and sputums became and remained negative for tubercle bacilli. We were unsuccessful in locating a small 1.5 to 2 cm. cavity in each of two patients; the sputum of one of these patients became negative after the procedure. During the sixth incomplete operation the patient died of cerebral air embolus following needle exploration of the cavity.

We thus performed a single (or staged) operation on sixty patients, while fourteen had multiple or incomplete operations. The eighty-four procedures were distributed among the seventy-four patients as shown in Table V.

RESULTS

The good results of almost any therapeutic measure for tuberculosis vary inversely with the activity and extent of the disease, while the complications are likely to be directly proportional to the degree of tuberculous involvement. These relations were encountered in the outcome of cavernostomy operation on the four different groups of patients in this series.

General Results.—In evaluating the results of cavernostomy operations, we listed patients as apparently arrested when the wound was healed without drainage, the cavity remained closed in serial x-ray examinations, and repeated examinations of smears of pooled, concentrated sputum were negative for tubercle bacilli for a minimum of eight months and continuously up to April 1, 1946, with no active lesions in the opposite lung.

Patients were classed as improved,* if the wound was contracting about the drainage tube or reduced to a sinus too small for a tube; the cavity by x-ray was invisible or no larger than the drainage tube, and sputum examina-

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*One patient is listed as improved who had cavernostomy for a large lower lobe cavity with a pre-existent contralateral cavity under a thoracoplasty collapse. The cavity which was drained has disappeared and the wound is solidly healed. We think it possible to close the cavity in the opposite lung by revision thoracoplasty.

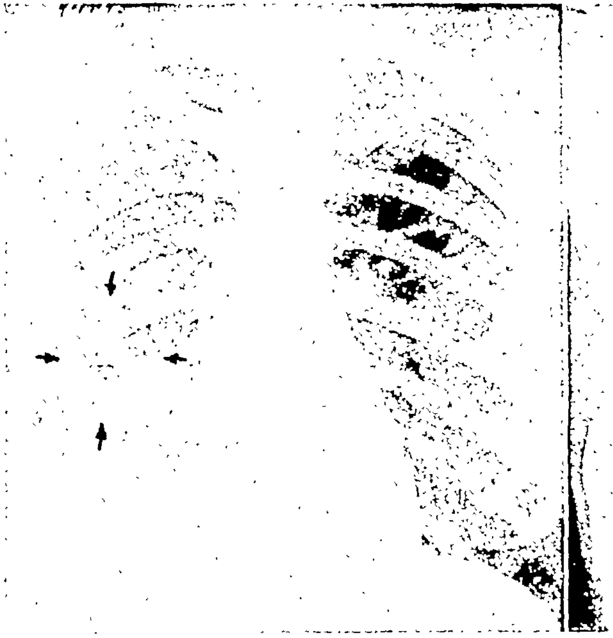

Fig. 1.

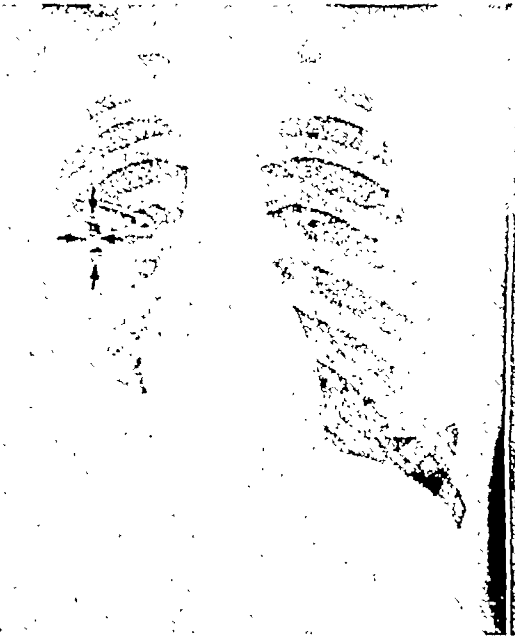

Fig. 2.

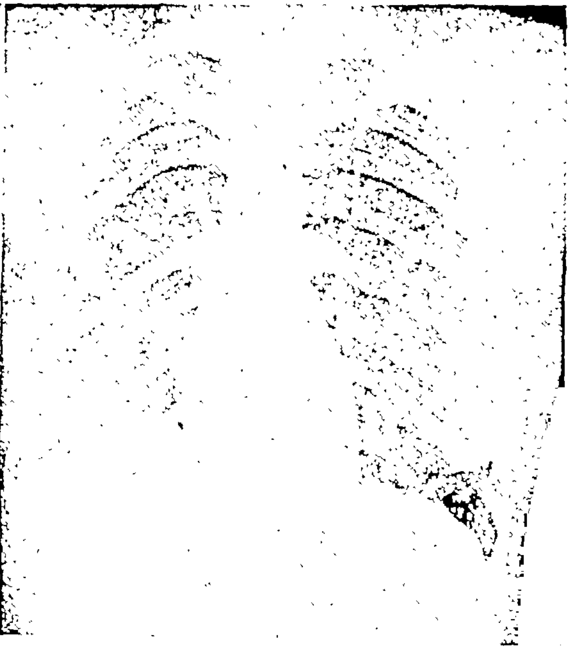

Fig. 3.

Fig. 1.—Case 1, L. W., April 17, 1943. There is a 4 cm. cavity with a fluid level in the right lower lobe posteriorly. The right leaf of the diaphragm is elevated following phrenic nerve interruption; previous pneumothorax was unsuccessful. There is a metal marker[20] on the skin. A few exudative infiltrations and a calcified lesion are present in the left midlung field.

Fig. 2.—Case 1, L. W., Sept. 29, 1943. The cavity in the right lower lobe is smaller, and a drainage tube is in the cavity. The exudative lesions in the left lung are clearing three months after cavernostomy, which was performed in June, 1943.

Fig. 3.—Case 1, L. W., May 10, 1944. This film was taken prior to discharge. No cavity is visible in either lung. The surgical defect in the right seventh rib is partially rebridged by new bone. Disease in the left lung appears stable. Sputum is repeatedly negative for tubercle bacilli on concentration and culture of pooled specimens.

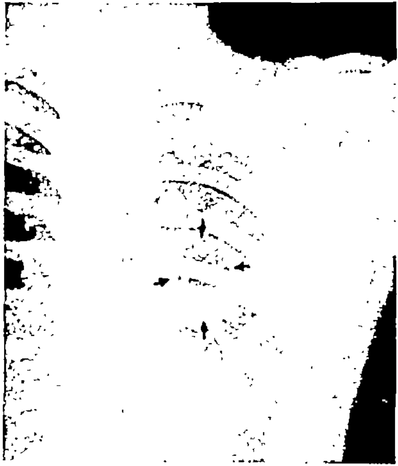

Fig. 4.

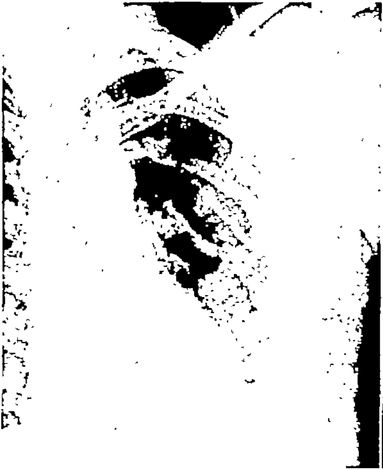

Fig. 5.

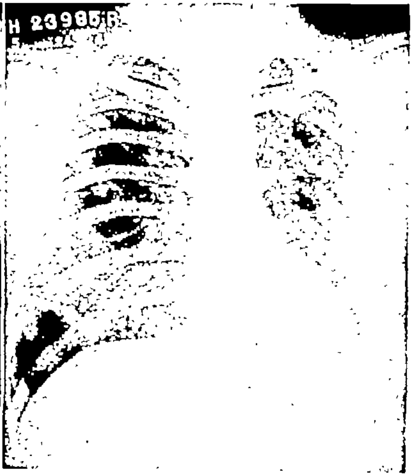

Fig. 6.

Fig. 4.—Case 2, W. McC., Nov. 1, 1943. A 7 cm. cavity is present near the left hilum in the lower lobe, a difficult location for closure by collapse therapy. A fluid level is present in the cavity, and two metal markers are placed on the skin anteriorly and posteriorly. The left leaf of the diaphragm is elevated and paralyzed. Left pneumothorax was unsuccessful.

Fig. 5.—Case 2, W. McC., July 18, 1944. The cavity is no longer visible in the left lung. At operation on Nov. 4, 1943, lengths of several ribs were removed and the cavity was opened and packed. No drainage tube was used; the cavity was treated by daily packing until it closed two months later. The resected ribs show partial regeneration in this film.

Fig. 6.—Case 2, W. McC., May 21, 1945. This film was taken prior to discharge from the hospital. No cavity is visible in either lung. Sputum is repeatedly negative for tubercle bacilli by concentrate and culture. The wound remains solidly healed.

Fig. 7.

Fig. 8.

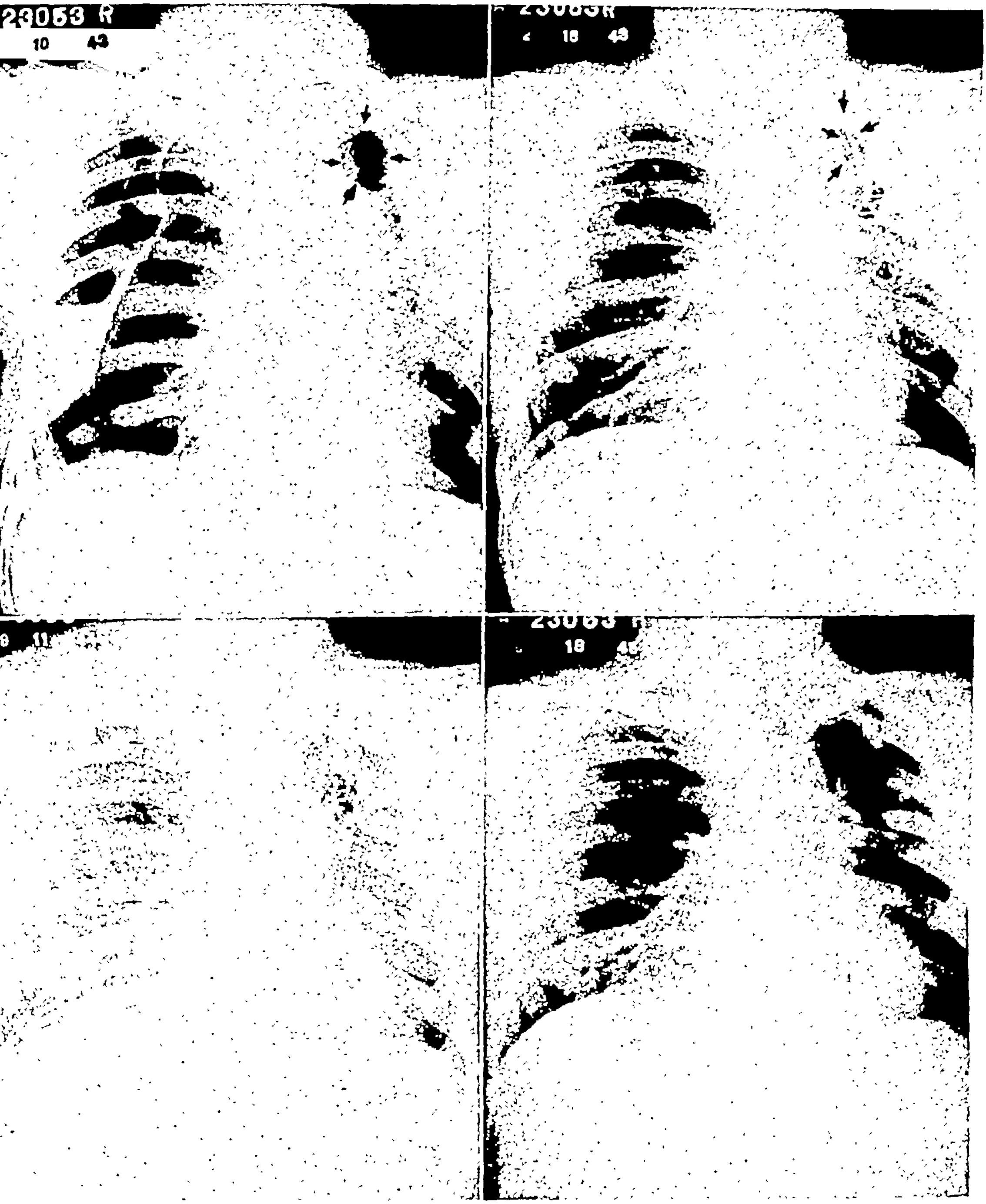

Fig. 9.

Fig. 10.

Figs. 7, 8, 9, and 10.—(*For legends see opposite page.*)

tions as described remained negative. If sputum examinations remained occasionally positive, or if the cavity was still visible in the roentgenogram, or if wound healing was unsatisfactory, the patient was considered unimproved. Those patients who were operated on in the past eight months are listed as too early for classification.

Of the entire group, thirty patients are apparently arrested or improved. Eighteen of these are at home, of whom four have intermittent slight serous drainage from a small sinus. Five who have cavity closure, healed wounds, and negative sputum, are soon to be discharged from hospital care. Six more with negative sputum and no definite cavity still have a small draining wound sinus, with examinations of the drainage negative for tubercle bacilli. One with cavity closure and a well-healed wound will need revision thoracoplasty for a residual cavity in the opposite lung.

Eight patients are unimproved, one of whom is worse. Six operated on since August, 1945, are not classified, although two now have negative sputum examinations. Thirty are dead. These results are classified in Table VI according to the original indication for cavernostomy.

TABLE VI. RESULTS OF CAVERNOSTOMY

GROUP	INDICATION	NO. PATIENTS	APPARENTLY ARRESTED (%)	IMPROVED (%)	UNIMPROVED (%)	NOT CLASSIFIED (%)	(%) DEAD
I	Low cardiorespiratory reserve						
	A. Definite dyspnea	26	4	12	8	15	61
	B. No dyspnea at rest	15	33	27	7	7	26
	Total	41	15	17	7	12	49
II	Residual cavity after thoracoplasty	11	55	9	-	9	27
III	Lower lobe cavity	6	83	-	17	-	-
IV	Other conditions	10	-	20	20	-	60
	Operation incomplete	6	50	-	33	-	17
	Total patients	74	27	13	11	9	40

It is apparent that the best results from the procedure were obtained in the group with residual cavity after thoracoplasty, in which 55 per cent are apparently arrested, and 9 per cent improved; and in the patients with lower lobe cavity, of whom 83 per cent are apparently arrested. The least favorable results and the highest mortality rate occurred when cavernostomy was used as

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Fig. 7.—Case 3, E. G., Sept. 10, 1943. There is a partial collapse of the right lung by pneumothorax, with an acute pleural effusion which became empyematous. The upper two-thirds of the left lung show exudative infiltrations, with a 6 cm. cavity in the apex of the upper lobe posteriorly. This patient was extremely ill, with a high fever and rapid loss of weight. The former plan for a left thoracoplasty had to be abandoned because of dyspnea.

Fig. 8.—Case 3, E. G., Dec. 10, 1943. The right empyema has almost disappeared; it was treated by frequent, repeated aspirations. On the left, the apical cavity is much smaller after cavernostomy, and a rubber drainage tube is in place in the cavity.

Fig. 9.—Case 3, E. G., Sept. 11, 1944. The right pyopneumothorax has been completely re-expanded. Minimal pleural thickening is visible below the right clavicle. On the left, the cavernostomy tube has been removed, the wound is completely healed, and no cavity is visible. Contrast shadows are caused by irregular rib regeneration on the left. Sputum examinations are negative.

Fig. 10.—Case 3, E. G., June 18, 1945. This film was made just before discharge. No cavity is visible on either side. There is a soft-tissue defect on the left posterior thorax. The patient is asymptomatic, and sputum is repeatedly negative for tubercle bacilli by smear and culture of pooled specimens.

Fig. 11.

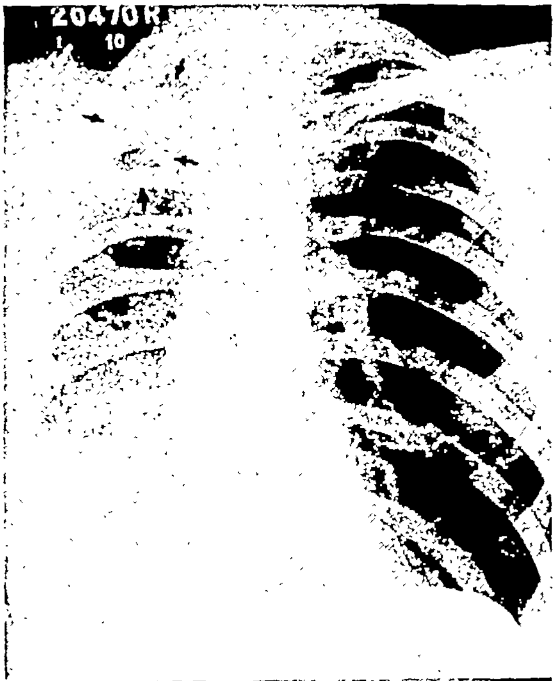
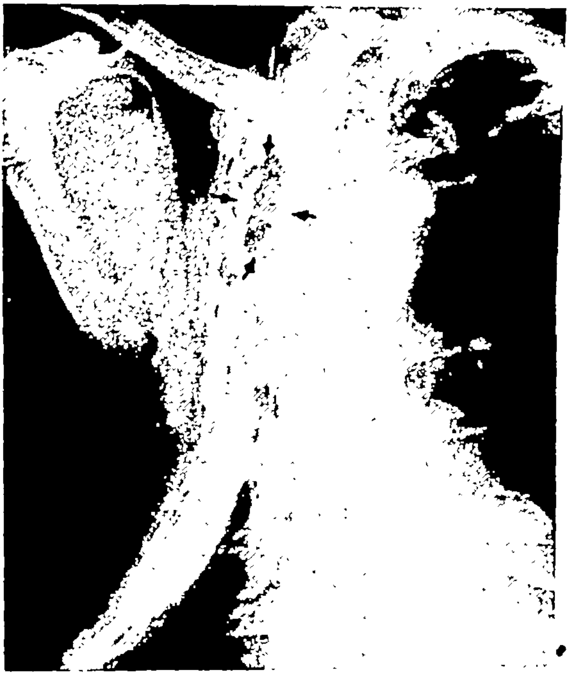


Fig. 12.



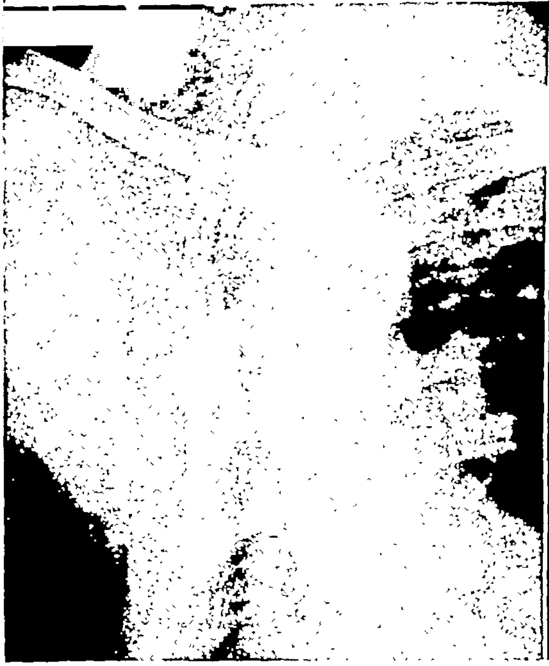


Fig. 13.

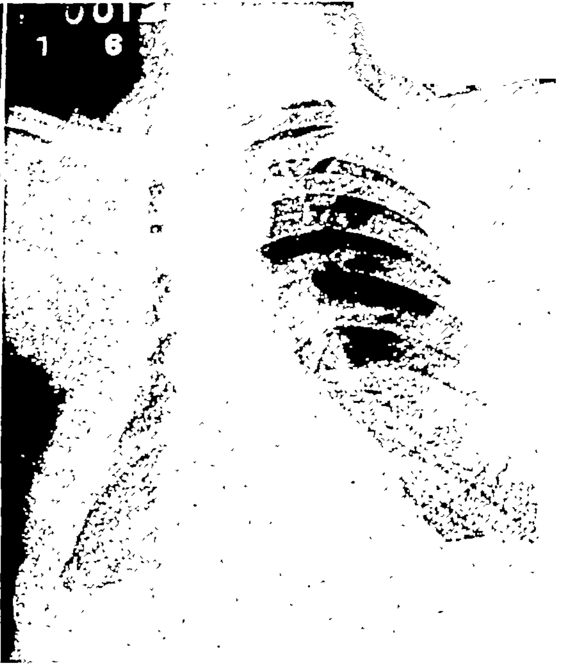


Fig. 14.

Figs. 11, 12, 13, and 14.—(*For legends see opposite page.*)

a salvage measure of last resort. *Among the patients with inadequate cardiorespiratory reserve*, 15 per cent are arrested and 17 per cent improved; 49 per cent expired. Of the miscellaneous patients with active or progressive tuberculosis, none was arrested and only 20 per cent are improved; the deaths amounted to 60 per cent.

The variation in the operative results is also shown strikingly when these patients are reclassified according to the apparent surgical risk at the time of cavernostomy. Most of those with residual cavity after thoracoplasty (Table I, Group II), or with a lower lobe cavity (Table I, Group III), were in fairly good condition for surgery, although three had exertional dyspnea and one was an elderly man nearly 70 years of age. In general, they had adequate pulmonary function and quiescent tuberculous lesions (apart from the cavity drained). They may be termed "good risks." Of these seventeen individuals, 65 per cent are arrested and 6 per cent improved; 17 per cent are dead. The fifteen patients in Group I-B (Table I) with no visible dyspnea had bilateral tuberculous lesions with obvious impaired function of the opposite lung. Most of them, however, did not have active contralateral disease at the time of surgery, and could be considered "fair risks" for cavernostomy. They appeared wholly unfit for thoracoplasty or resection, since our observations showed most of the pulmonary function lay in the lung operated upon. Thirty-three per cent of these patients are arrested, 27 per cent are improved, and 27 per cent are dead.

TABLE VII. RESULTS OF CAVERNOSTOMY

SURGICAL RISK	NO. PATIENTS	APPARENTLY ARRESTED (%)	IMPROVED (%)	UNIMPROVED (%)	NOT CLASSIFIED (%)	DEAD (%)
Poor risk	36	3	14	11	11	61
Fair risk	15	33	27	6	6	27
Good risk	17	65	6	0	6	17
Total patients	*68*	*27*	*13*	*11*	*9*	*40*

The remaining thirty-six patients in Groups I-A and IV (Table I), excluding those six in whom cavity drainage was not accomplished, had definite dyspnea, or progressive tuberculosis, or both, at the time of operation. They were undoubtedly "poor risks" for any form of therapy. Those now dead amount to 61 per cent; 14 per cent are improved, and only 3 per cent apparently arrested. These figures are summarized in Table VII.

Fig. 11.—Case 4, W. C., Jan. 10, 1941. A 7 cm. cavity is present in the upper third of the right lung, with scattered infiltrations throughout the remainder of the lung. The left lung is clear. The right apical cavity remained open after pneumothorax and partial pneumonolysis. Persistent pleural effusion appeared later and the pneumothorax was re-expanded.

Fig. 12.—Case 4, W. C., April 17, 1942. This film was made after patient had had seven stages of right thoracoplasty including three revision operations. In all, nine ribs have been resected. A 4 cm. cavity is still present below the right clavicle, and sputum is persistently positive for tubercle bacilli.

Fig. 13.—Case 4, W. C., April 6, 1944. Right cavernostomy was done Sept. 12, 1942, and the cavity was opened through the posterior thoracoplasty scar. The wound is now completely healed and the cavity closed ten months after open drainage. This film shows irregular contrast shadows which are due to the soft-tissue defect in the posterior thorax. Sputum is negative for tubercle bacilli by smear and culture of pooled specimens, and the patient is symptom-free.

Fig. 14.—Case 4, W. C., July 6, 1945. The patient was discharged from the hospital more than a year previously. He has been gainfully employed for the past eight months. Sputum examinations are negative for tubercle bacilli.

Wound Healing.—Since a number of the operations were of too recent a date to allow judgment of the final result, those patients who were operated on more than one year ago, before April 1, 1945, were studied separately, to ascertain the fate of the wound, the cavity, and the related ipsilateral tuberculous lesions. Only a few of the living in this group had previous active disease on the opposite side.

Table VIII classifies the wound healing of all thirty-two living patients with cavernostomy done more than a year ago. Among these the cavity is closed and the wound is healed in eighteen (56 per cent of the living patients), and such lesions are here considered healed. Six more (19 per cent) are classed as healing, with the cavity apparently closed, or small and indefinite; and the wound reduced to a small, draining sinus. Eight (25 per cent of the living patients) are unhealed, with open cavity and a drainage tube in place in the surgical sinus.

TABLE VIII. WOUND HEALING AFTER CAVERNOSTOMY (LIVING PATIENTS OPERATED ON BEFORE APRIL 1, 1945)

			STATUS OF WOUND		
GROUP	INDICATION FOR OPERATION	NO. PATIENTS	HEALED (%)	HEALING (SMALL SINUS) (%)	UNHEALED (DRAINAGE TUBE IN PLACE) (%)
I	Low cardiorespiratory reserve				
	A. Definite dyspnea	6	50	-	50
	B. No dyspnea at rest	9	45	33	22
	Total	15	47	20	33
II	Residual cavity after thoracoplasty	8	75	25	-
III	Lower lobe cavity	6	83	-	17
IV	Other conditions	3	-	33	67
	Total patients	32	56	19	25

The appearance of the wound postoperatively, and the rate of wound healing were both surprisingly good in most instances, and often exceeded our expectations. Final healing was, of course, slower than in a nontuberculous lesion, but only six wounds, after several weeks, still showed the indolent, supposedly characteristic, tuberculous appearance with absence of granulation tissue and marginal undermining. By far the greater number had early development of firm pink granulations, with progressive epithelization of the edges. We frequently had to dilate the sinus gently to maintain an adequate stoma until the bronchial fistula closed. As mentioned before, several of the wounds healed prematurely in spite of such efforts.

Among the apparently arrested patients, healing was considered complete on the date that the wound or sinus became completely and permanently covered with epithelium, with no further evidence of drainage. The time required for wound healing ranged from five to twenty-one months in all cases except for one minute but persistent sinus which required forty months before the slight intermittent drainage ceased. The average time was slightly over twelve months.

TABLE IX. SPUTUM CONVERSION AFTER CAVERNOSTOMY (LIVING PATIENTS OPERATED ON BEFORE APRIL 1, 1945)

GROUP	INDICATION FOR OPERATION	NO. PATIENTS	POSTOPERATIVE SPUTUM EXAMINATION	
			NEGATIVE (%)	POSITIVE (%)
I	Low cardiorespiratory reserve			
	A. Definite dyspnea	6	67	33
	B. No dyspnea at rest	9	89	11
	Total	15	80	20
II	Residual cavity after thoracoplasty	8	75	25*
III	Lower lobe cavity	6	83	17
IV	Other conditions	3	67	33
	Total patients	32	78	22

*Two patients in whom cavernostomy was not completed and the cavity was never opened.

TABLE X. POSTOPERATIVE COMPLICATIONS (NONFATAL)

COMPLICATION	NO. PATIENTS
Traumatic pleural effusion	3
Traumatic pneumothorax (operative)	2
Increased dyspnea	8
Late hemorrhage from wound (one week to one year)	8
Air embolus (immediate)	2
Air embolus (one year later)	1
Infection of chest wall	1
Iodoform reaction (from gauze packing)	1
Loss of gauze in wound (after a dressing one year postoperative)	1
Tuberculosis of spine	1
Wound healing over residual cavity	1
Reopening of cavity after wound healing	6
Total complications	35

TABLE XI. CAUSES OF DEATH (LESS THAN THREE MONTHS POSTOPERATIVELY)

CAUSE OF DEATH—EARLY	NO. PATIENTS
Hemorrhage from cavity or wound	2
Air embolus, operative	2
Respiratory failure	5
Exacerbation of tuberculosis	2
Auricular fibrillation and cardiac decompensation	1
Pulmonary embolus	1
Empyema and bronchopleural fistula	1
Peritonitis (perforation of tuberculous ileitis)	1
Total early deaths	15

TABLE XII. CAUSES OF DEATH (MORE THAN THREE MONTHS POSTOPERATIVELY)

CAUSE OF DEATH—LATE	NO. PATIENTS
Progression or exacerbation of tuberculosis	11
Accidents from postoperative dressing	
A. Traumatic pneumothorax with bronchopleural fistula	1
B. Air embolus	1
Hemorrhage from cavity	1
Lobar pneumonia and cor pulmonale	1
Total late deaths	15

The three wounds resulting from generous rib resection about a lower lobe cavity in good-risk patients showed slight initial delay in healing. Final closure and cessation of drainage from the saucerized defect, however, occurred earlier than in many wounds in which a narrow, deep sinus persisted. In most of the patients with a pre-existent thoracoplasty collapse, the cavernostomy wound healed rather rapidly.

Sputum Conversion.—A prompt conversion of positive sputum to negative was a very common occurrence after cavernostomy, as has been reported following the Monaldi procedure of cavity aspiration. Bacteriologic examination of the purulent or serous drainage from the wound was not done routinely, because this was also nearly always found to be negative for tubercle bacilli. Sputum conversion did not occur in those patients with progressive tuberculosis or with exacerbations of disease after surgery, nor, of course, in those few with open cavity on the opposite side (see Table IX).

Postoperative Complications and Deaths.—It has been seen that nearly 60 per cent of these operations were performed upon patients with a low cardiorespiratory reserve. A considerable number had obvious dyspnea. In several patients, there were other active or progressive tuberculous lesions in addition to the cavity subjected to drainage. Nearly all had undergone previous, unsuccessful collapse therapy. The potential complications from an operation of this type upon such patients are obvious and scarcely require detailed discussion. The nonfatal complications are listed in Table X, and the causes of death, early and late, in Tables XI and XII.

It is, however, worthy of comment that most of the deaths may be attributed to evolution of the previous tuberculous disease. Respiratory failure (five patients), hemorrhage from the cavity (four patients), and progression or exacerbation of tuberculosis (thirteen patients) account for 73 per cent of the deaths. Mention may also be made of the effect of cavernostomy upon the ventilatory efficiency of the patients. There would be presumptive danger from the surgical creation of a bronchocutaneous fistula in a person whose respiratory mechanism was sufficiently disturbed to show dyspnea at bed rest. A notable increase in dyspnea was, perhaps, not frequent in relation to the poor condition of many of these patients. Nevertheless, it was noted thirteen times, and was five times the chief cause of death. We believe this possibility should be considered when employing cavernostomy on a patient with clinical dyspnea.

SUMMARY

1. A report is presented of the indications, operative technique, surgical complications, and results of eighty-four cavernostomy operations upon seventy-four patients.
2. The more usual methods of collapse therapy had been previously employed on nearly all of these patients.
3. The patients are divided into four groups according to the indication for operation.
4. Forty-three patients with low cardiorespiratory reserve (Group I) had reduced function of the other lung which precluded collapse therapy or pul-

monary resection. Of these, twenty-six had clinical dyspnea and seventeen had none. After cavernostomy, thirteen (32 per cent) are apparently arrested or improved; three (7 per cent) are unimproved; five (12 per cent) are too early for classification; twenty (49 per cent) are dead. Two others in this group had an incomplete operation without drainage of the cavity; both are now apparently arrested. The presence of dyspnea seemed an important factor militating against the success of cavernostomy. Among the patients *without* dyspnea (but with low cardiorespiratory reserve) five (33 per cent) are apparently arrested and four (27 per cent) improved, with four (27 per cent) dead. Among the patients *with* visible dyspnea at rest, only one (4 per cent) is apparently arrested and three (12 per cent) improved, with sixteen (61 per cent) dead.

5. Thirteen patients (Group II) had a residual cavity beneath a thoracoplasty collapse. Six of these (55 per cent) are apparently arrested, and one (9 per cent) improved. One (9 per cent) is too early for classification and three (27 per cent) are dead. Two others in this group had an incomplete operation and are unimproved.

6. A large lower lobe cavity not closed by pneumothorax or phrenic paralysis or both, was present in eight patients (Group III). There was no important tuberculosis in the opposite lung. Five (83 per cent) are arrested, and one (17 per cent) is unimproved. Cavity drainage was attempted but not achieved in two others; one is now apparently arrested and one is dead.

7. Cavernostomy was done upon ten patients (Group IV) with active or progressive tuberculosis and miscellaneous indications for surgery. Five of these had previous unsuccessful Monaldi intercostal drainage which was converted to cavernostomy. Two patients (20 per cent) are improved; two (20 per cent) are unimproved; and six (60 per cent) are dead.

8. The sixty-eight patients whose cavities were successfully drained were also classified as good, fair, and poor risks for surgery, according to their pulmonary function and extent of active tuberculosis. It was shown that the fair-risk and poor-risk patients could not have withstood collapse therapy or pulmonary resection because of reduced function in the contralateral lung. Among seventeen good-risk patients, eleven (65 per cent) are apparently arrested and one (6 per cent) is improved after cavernostomy. One each (6 per cent) is unimproved and too early for classification, and three (17 per cent) are dead. Out of fifteen fair-risk patients, five (33 per cent) are apparently arrested and four (27 per cent) are improved. One each (6 per cent) is unimproved and too early for classification. Four (27 per cent) are dead. The patients who were poor risks for cavernostomy and unsuitable for any other surgery numbered thirty-six. One (3 per cent) is apparently arrested and five (14 per cent) improved. Four each (11 per cent) are unimproved and too early for classification. Twenty-two (61 per cent) are dead.

9. Most of the postoperative complications and deaths occurred in patients who before operation had visible dyspnea at rest, or active, progressive tuberculosis. Nearly 75 per cent of the deaths were attributable to these two factors.

10. The wounds which healed most readily were those of post-thoracoplasty cavities, and lower lobe cavities with generous rib resection and some degree of surgical collapse in the area of the cavity.

CONCLUSIONS

1. When the usual measures of collapse therapy are ineffective for cavity closure, cavernostomy is frequently successfully used for residual cavities after thoracoplasty. Of eleven patients with such cavities, 55 per cent became apparently arrested with cavity closed, wound healed, and sputum negative for tubercle bacilli. An additional 9 per cent was improved with negative sputum, wound reduced to a small sinus, and cavity closed or not definitely visible.

2. Likewise, after an unsuccessful trial of the usual procedures of collapse therapy, cavernostomy is usually effective in closing lower lobe cavities, in patients with adequate cardiorespiratory reserve. Of six such patients, 83 per cent became apparently arrested.

3. Cavernostomy may be a useful procedure for patients *without* dyspnea or progressive tuberculosis, in whom reduced function of the opposite lung prohibits collapse therapy or pulmonary resection. Open cavity drainage produced apparent arrest in 33 per cent, and improvement in 27 per cent of fifteen patients in this group.

4. Cavernostomy yields few good results in patients with obvious dyspnea or progressive tuberculosis. In these instances, the operation was followed by apparent arrest in 3 per cent and improvement in 14 per cent of thirty-six patients.

5. Healing of the wound and the cavity appears to be most prompt when cavernostomy was preceded or accompanied by some surgical collapse of the involved pulmonary tissue.

6. Most of the deaths and serious complications occurred in patients who had poorly resisted advancing tuberculosis, or who were visibly short of breath prior to cavernostomy.

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DISCUSSION

DR. W. A. HUDSON, Detroit, Mich.—I want to agree very heartily with what Dr. Test has just presented. I think there are definite indications for the use of cavernostomy, but they are limited indications. In general, we may use cavernostomy to make our patients more comfortable. We may use it to save a life or prolong a life. I think those are the only three indications we have.

Dr. Test has gone into more detail as to how we can select the patients. I differ somewhat from Dr. Test's and Dr. O'Brien's group in approach to the cavernostomy. As far as possible, I like to clean up the cavern before doing the cavernostomy. Frequently we still insert a tube, apply drainage and suction, merely as a preparatory measure, thereby ridding the cavity of much of the secretion that may be present when it is open. We reduce what Dr. Archibald used to call "marshy lung" about the cavities.

In addition, I notice that Dr. O'Brien and his associates seldom use skin flaps. I have found skin flaps very helpful in making the patient more comfortable during the convalescent period, especially at the time you are doing dressings, and it saves many secondary operations.

These are slides of the type of skin flaps that are used. These examples range from a period of four years to nine years since cavernostomy.

(Slide.) This is very soon after the opening of the cavern. You will note that an anterior approach has been used, and there has been a skin flap to line the upper or proximal portion of the cavity. There has been a skin flap from the anterior lateral chest wall to line the anterior lateral portion of the cavity and a short flap to line that portion of the cavity which lies directly lateral, leaving only one raw surface which is already undergoing healing.

(Slide.) Here is a patient who is almost healed; there is a small sinus which is readily dressed. There is a bronchial opening of considerable size in the bottom of that cavity, and as long as the fistula is present I think it very improper to allow the external opening to close. For that reason a skin flap has proved very effective.

(Slide.) Here is another cavity which is lined with skin down to and including part of the base of the cavity. This portion has been covered and lined by epithelium growing out from the bronchial tree. It is now six years since the cavernostomy.

(Slide.) These patients are all free of sputum, and are going about their daily activities. This case demonstrates depth of the cavity and the small residue of visible lung in the depth of the cavity.

(Slide.) This shows a very extensive cavity. The patient was operated upon nine years ago, after he had been rejected for all other forms of therapy, or had failed to get a result from the procedures tried. There is an extensive cavity extending from the apex to midlung field and in contact with chest wall. We first established drainage by suction, cleaned up the cavity, relieving the patient of an aggravating cough, and then proceeded with cavernostomy, which converted the large cavity into a pleurocutaneous cavity. What we have really done is to exteriorize the cavity.

This patient, for a period of about fourteen years, had lived at the expense of Wayne County, but during the past few years he has supported himself.

(Slide.) This shows the extent of the scar in the wall of the cavity about one inch in thickness and the small amount of lung that was resected. We tried to do a very generous resection of the outer wall of the cavity, and we feel that it is very important to remove as much of the parietal wall as possible. The flaps are placed so that they will not be under tension, and so there will not be too much difficulty in inserting gauze for dressing and pressure.

DR. W. L. ROGERS, San Francisco, Calif.—I only rise for two or three comments, mainly because Dr. Shipman and I presented a paper before this organization at the Toronto meeting on this subject.

It is somewhat doubtful whether or not one is justified in selecting this procedure as a primary measure except in a very selected, small group where the cavity lies posteriorly in the apex of the lower lobe, where it has not responded to other reversible forms of collapse therapy, and where, by cavernostomy, conversion may reasonably be expected.

The only other specific indication for this procedure is in a small percentage of residual cavities underlying apical thoracoplasties, and I believe in that group where we may hope for sputum conversion, it is a justifiable secondary procedure. This indication has been covered by their Groups II and III.

All other indications I am very much in doubt about, and am afraid if we pursue cavernostomy with the broader indications, it will prove disappointing.

We feel that a two-stage operation is advisable. In two of our cases, after preparing the flap and resecting small sections of one or two ribs overlying the cavity, while waiting for the flap to become adherent, the underlying cavity closed, again illustrating the unpredictable nature of these cavities.

Second, we prefer not to pack the cavity after it is opened.

Third, we believe the skin flap is very essential, because many of the early failures which were performed many years ago were due to early stenosis of the wound with closure before the cavity had been converted and obliterated. It is true that a sinus may, at times, remain, but if sputum conversion has been obtained the sinus may be closed subsequently.

DR. F. C. TEST—Dr. Rogers' comment about the limited indications for this operation is very well taken. As you see, some of our best results were obtained on residual cavities after an apical thoracoplasty. We feel, however, that the high percentage of favorable results after drainage of lower lobe cavities may warrant further trial of cavernostomy for basal cavity when routine collapse therapy has proved ineffective.

The comments about the use of a skin flap were interesting. A constant sequel to cavity drainage is the persistence of a chronic open sinus over many months. Skin flaps may be the answer, but we have intentionally not used them hoping to avoid creating a permanent bronchocutaneous fistula. We were rewarded by obtaining a number of firmly healed wounds after the procedure.

CARDIAC RESUSCITATION

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THE management of cardiac standstill and ventricular fibrillation during a surgical operation presents many complex problems. A set of rules and procedures which in some instances can save life has been established gradually due to the experience acquired in both the experimental and clinical fields. These rules and procedures have been amply described in the literature. They will not, therefore, be recalled here.

In this paper, we will confine the discussion to (1) the essential characteristics of cardiac resuscitation, (2) the rarity of restoration of cardiac activity *following cardiac standstill and ventricular fibrillation in clinical cases, and* (3) the presentation of a few procedures which can be of great assistance to control a few important causes of failure in cardiac resuscitation.

This report is based on the experience acquired experimentally during the past three years. More than 200 experiments were made on the dog's heart. Cardiac standstill or ventricular fibrillation was induced by various means, altering seriously the myocardial structure and functions, or they were the spontaneous result of conditions created during operation on the heart. In every experiment, electrocardiograms as well as blood pressure changes were recorded with many other findings of interest and value. It seems possible to state that now most of the dogs presenting spontaneous cardiac standstill or ventricular fibrillation during operation are definitely revived. These encouraging results do not let us forget, however, that much work has yet to be done in the experimental field before one can control systematically the numerous factors which at one moment or another can prevent cardiac resuscitation.

DEFINITION OF CARDIAC RESUSCITATION

Cardiac resuscitation means full and durable restoration, in due time to avoid eventual death, of the essential functions of a heart which has lost its power to propel blood, and which, in all appearances, is unable to recover spontaneously its effective value as a pump.

This definition, imperfect as it may be, has the advantage, it is believed, of embodying the essential features of real cardiac resuscitation and of limiting the use of the term to its strict and true sense. First, *the heart must have stopped functioning as a pump.* Many hearts are "resuscitated" which are still beating with a certain degree of circulatory efficiency. Second, *cardiac functions must be fully and permanently restored.* This signifies that resump-

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tion of coordinated beats without concomitant recovery of adequate propulsion of blood to the periphery as well as temporary revivals of the cardiac beat and circulation for only short periods of time should not be considered resuscitation. Third, *life must be preserved.* This implies that fully operating cardiac functions are not sufficient. Serious and permanent damage to vital centers or organs due either to transcending the limit of resuscitability or improper technique should be avoided. It is, indeed, a well-known fact that resuscitation of any given organ is inextricably dependent on the maintenance of the functional ability of the others. The heart is no exception to that rule.

If one accepts the fulfillment of these three conditions for true cardiac resuscitation, it soon becomes obvious that cases of real cardiac revival are not as numerous as the literature tends to show. Cardiac resuscitation following ventricular stoppage of more than a few minutes is a rare event clinically. On the other hand, it is more than probable that not a single case of ventricular fibrillation in the human has yet been revived in the sense mentioned above.

BRIEF DISCUSSION OF CARDIAC RESUSCITATION CASES REPORTED IN THE LITERATURE

A. *Resuscitation of Cardiac Standstill.*—It is well known that when the heart ceases to propel blood, a series of phenomena develops leading rapidly to death. The aortic and pulmonary circulations fail. The systemic and coronary pressures fall to zero. Anemia of the brain and spinal cord follows immediately with ultimately fatal effects. Richet[1] found that three minutes is a sufficient interval to cause death of the medulla deprived of blood in dogs. Stewart and associates[2] have shown that seven minutes is approximately the average limit of complete anemia tolerated by the nervous system, and that it is rarely extended to fifteen minutes. Weinberger and associates[3] found that when the circulation is interrupted for eight minutes and forty-five seconds or longer, life could not be restored for more than a few hours.

The clinical reports of resuscitation published, where cardiac arrest greatly exceeded the limit of resuscitability found by experimenters, should not be regarded as evidence against experimental demonstration. The latter establishes its conclusions in much more favorable and accurate conditions. Contrarily, many sources of errors are unavoidable in clinical interpretation of cardiac arrest. The time elapsing from standstill to re-establishment of cardiac activity in a patient with an unopened chest is not easily recorded. Indeed, the inception of ventricular stoppage is impossible to determine except by actual inspection. Even electrocardiographic tracings cannot help in this occurrence.

B. *Resuscitation of Ventricular Fibrillation.*—Very few attempts at cardiac resuscitation following ventricular fibrillation during operation have been reported in the literature. We believe that all of them ended in failure.

Beck[4] reported two cases of defibrillation of the human heart. The patients were not revived.

Barber and Madden[5] reported a patient who, according to them, showed ventricular fibrillation and was temporarily revived. No electrocardiograms were taken. Therefore, one cannot discard the possibility that paroxysmal ventricular tachycardia or ventricular flutter may have been confused with fibrillation. A fact that strongly suggests that this is probably what happened is that a normal cardiac rhythm was restored by a method which in experimental conditions is never effective to resuscitate the fibrillating heart, at least in the dog.

Thompson and associates[6] reported a case of "successful resuscitation" with operative recovery of the patient following cardiac cessation due to auricular and ventricular fibrillation. No electrocardiograms accompany their report to establish firmly the accuracy of their clinical interpretation. They massaged the heart, and injected intravenously 2 c.c. of neosynephrin, and 1 c.c. of nikethamide. Within one minute after the injection, the heart was definitely revived. They suggest that resuscitation may have been due entirely to cardiac massage and pulmonary ventilation.

As far as we know, not a single case of ventricular fibrillation in dogs or in patients has been revived only by cardiac massage or pulmonary ventilation with or without drugs similar to those used in their case. Pulmonary ventilation per se or combined with massage is not able to annihilate ventricular fibrillation. Massage stimulates the myocardium. It tends, therefore, to increase the force and the continuation of fibrillary movements. It never has the effect of rendering quiescent fibrillating myocardial fibers and is not used in cardiac resuscitation attempts for that purpose. Every fibrillary movement must be eliminated before the fibrillating heart can resume a coordinated rhythm. This essential rule in defibrillation has been emphasized by Wiggers and confirmed by every experimenter with sufficient experience with ventricular fibrillation.

In the case of Thompson and associates, it is important to remember that auricular fibrillation preceded the ventricular rhythm they describe as ventricular fibrillation. In auricular fibrillation, it is not rare to see an irregular, rapid and bizarre ventricular rhythm, which is not fibrillation, appearing and disappearing *spontaneously* after a variable time even while the auricles continue to fibrillate. This is a frequent observation in cardiac experimental work when the auricles are mechanically stimulated, causing auricular fibrillation. As early as 1894, François-Franck[7] showed that auricular fibrillation sometimes can determine this rapid and bizarre ventricular rhythm. In 1903, he stated concerning the effect of auricular stimulation on the ventricles: "Une excitation assez forte, ne dépassant pas cependant la limite de tolérance du coeur, produit une tachycardia arythmique des deux ventricules, caractérisée par un état soutenu de resserement du myocardie qui constitue un véritable tétanos atténué à secousses dissociées et irregúlière."[8] Frédéricq[9] described this abnormal ventricular rhythm under the name of "rythme affolé des ventricules dû à la fibrillation des oreillettes." It is more than probable that Thompson and his co-workers took for ventricular fibrillation itself that bizarre, ventricular rhythm which sometimes accompanies auricular fibrillation.

On one occasion we had the opportunity to observe ventricular fibrillation during operation. The chest had just been opened and the heart exposed when the ventricles began to beat very feebly and the blood pressure dropped. The heart was massaged and 2 c.c. of epinephrin 1:1,000 were injected through the left auricular appendage. This cardiac stimulation provoked immediately ventricular fibrillation. Electrical shocks were administered to the myocardium. Coordinated beats followed immediately. Electrocardiograms were taken while heart was fibrillating, and after defibrillation. (Fig. 1.) The heart, however, never regained its value as an effective pump, in spite of medication. At autopsy, it was found that both coronary arteries were totally occluded by old sclerotic lesions at their aortic mouths. These lesions were undoubtedly sufficient to prevent cardiac resuscitation. Apparently no blood could enter the main coronary arteries during cardiac massage and the extracardiac collateral circulation was too poor to irrigate the heart properly. Adequate blood flow to the myocardium is essential in any attempt to revive the heart.

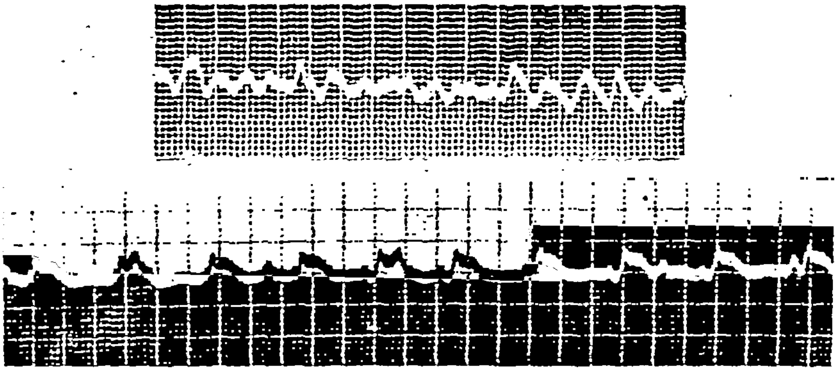

Fig. 1.—Electrocardiographic tracings from a patient who developed ventricular fibrillation during operation. Upper tracing during ventricular fibrillation, lower tracing after defibrillation.

In conclusion of this brief discussion about the clinical cases of cardiac resuscitation reported, we should like to reiterate that clinical interpretation of the dying heart's behavior and accurate timing of the onset of cardiac inaptitude to propel blood are not a simple matter. The difficulties inherent in this problem are a source of many errors exposing erroneous statements concerning cardiac resuscitation. Finally, one should not be deceived by some extraordinary reports in the literature concerning restoration of cardiac activity. Good results are exceptional. Failures are still numerous, even in the operating room where the conditions of success are unquestionably better than anywhere else.

Among the causes of failure in the operating room, loss of very precious time in trying at first simple and benign procedures as well as lack of organization and preparation to take care of any serious emergencies play an important role. These causes can and should be eliminated. There is no excuse for their persistence. With the essential equipment and a well-planned technique, a higher percentage of good results can be gained.

Other factors of failure which we have particularly studied experimentally are: (1) Hyperirritability of the heart, (2) absence of cardiac tone, (3) increase of the myocardial temperature, and (4) peripheral circulatory failure. It is believed that these conditions can be controlled to a large extent when recognized on time and treated properly.

SOME IMPORTANT CAUSES OF FAILURE IN CARDIAC RESUSCITATION

1. *Hyperirritability of the Heart.*—The development of cardiac hyperirritability in a nonfunctioning heart is due to (a) cardiac ischemic conditions always present in either cardiac standstill of a certain duration or ventricular fibrillation, and to (b) overstimulation of the myocardium by cardiac massage and intracardiac therapy during attempts to revive the heart. While the myocardial structure is hyperirritable, the slightest cardiac stimulation, mechanical or chemical, may suffice to transform a standstill condition into fibrillation or render useless methods usually efficient to resuscitate the heart from a fibrillary state.

It is, for instance, a well-known fact that in cardiac standstill intracardiac administration of drugs which in normal conditions only increase the strength and rate of the heart can provoke ventricular fibrillation. This is why intracardiac injection of epinephrin in patients with an unopened chest is rightly considered a dangerous method in cardiac resuscitation. These injections are made blindly, most of the time through the right or left ventricle instead of through the auricles, and their effects cannot immediately be controlled, except by electrocardiographic tracings. When they initiate fibrillation it is not known by the operator, and consequently no measures are taken to defibrillate the heart. Death ensues and the real cause of it remains unknown. On the other hand, it does happen that during attempts to resuscitate the heart from fibrillation, electrical shocks are successful in restoring a normal rhythm. Yet more or less rapidly, paroxysmal ventricular tachycardia develops, due to overstimulation of a hyperirritable heart, and fibrillation reappears. In such a state of affairs, repetition of electrical shocks seldom resuscitates the heart, which every time after defibrillation presents paroxysmal ventricular tachycardia leading to fibrillation.

Among the drugs to decrease cardiac hyperirritability rapidly and to eliminate, therefore, deleterious reflexes and dangerous ventricular rhythms, the best known are members of the cocaine group. Their usage for this purpose is far from being a new discovery in the physiologic field, as it has been stated in recent years.

François-Franck in his Leçons at the Collège de France from 1880 to 1904 stressed repeatedly the protective value of these drugs in cardiac experimental work. He recommended the application of cocaine hydrochloride solution on the surface of the heart and the great vessels at the base of the heart to avoid reflexes.[10] From an historic point of view, and to give credit where it really belongs, it is important to quote at length this great physiologist. In his leçons, during 1893, he says, concerning the technique of coronary arterial ligation, "ces opérations ont été rendues faciles, grâce à la cocainisation locale qui permet les manipulations de ces régions si impressionables, sans danger sérieux pour le coeur." Concerning the reflexes that one can provoke while working in the cardioaortic area, he remarks: "on observe tout particulièrement des réactions dans les irritations de la région sigmoidenne de l'aorte: elles disparaissent à la suite d'une cocainisation interstitielle localisée, pour reparaître quand la paralysie locale des réseaux sensitifs s'est dissipée.''[12] Other important studies on the same subject were published later on by Fauconnier,[13] Kochmann and Daels,[14] Gilbert,[15] Mathieu,[16] and Mathieu and Hermann,[17] to mention only a few. It became a routine procedure in many laboratories to use drugs of the cocaine group to reduce cardiac surface irritability. More recently, Mautz[18] carried out very interesting experimental work which confirms entirely the conclusions reached by many physiologists years ago on the value of surface application of the same drugs. He did not claim that the method he studied so well was something new.

These drugs also have been investigated as a measure to prevent ventricular fibrillation and as an aid in cardiac resuscitation from ventricular fibrillation. Both surface application and intravenous injection of these drugs were advocated years ago for the purpose of preventing ventricular fibrillation. The clinical usage of cocaine as an aid in cardiac resuscitation is unquestionably the logical application of a method long known to be valuable against cardiac hyperirritability. On the other hand the clinical use of electric shocks to defibrillate the heart has been the logical application of a method efficiently demonstrated by experimentation in the past century.

François-Franck studied the value of cocaine to increase the resistance of the heart against ventricular fibrillation caused by electrical shock. He says: ''Dans nos expériences comparatives, nous avons profité de l'action atténuante si évidente que provoque sur l'excitabilité cardiaque, la cocainisation générale du sujet (1 centigramme de cocaine par kilo, dose inférieure de moitié à la dose toxique); on obtient de même une tolérance remarquable du myocarde par l'application locale directe de cocaine. Dans ces deux conditions, telle excitation faradique, qui aurait sûrement tué le coeur normal, ne provoque qu'un tétanos dissocié, plus ou moins prolongé, à la suite duquel les ventricules se relâchent et reprennent instantanément leurs systoles et leurs diastoles normales.''[10]

Later on, summarizing the value of cocaine to reduce cardiac irritability in a lecture devoted to reactions following general or local administration of cocaine, he says: ''Sous l'influence de l'action locale de la cocaine, les manipulations directes du coeur, nécessaires à l'introduction d'appareils, ne risquent plus de provoquer les accidents de trémulation mortelle; l'irritation de la région sigmoidenne de l'aorte ne produit plus les accidents que nous avons décrits, et leur disparition prouve leur mécanisme réflexe; de même, l'insuffisance aortique, pratiquée à la suite d'une cocainisation interstitielle, se trouve réduite à ses effets mécaniques, et dégagée des réflexes vaso-constricteurs et autres que nous avons fait intervenir dans la pathogenié d'un certain nombre d'accidents accompagnant cette lésion. On peut aussi reprendre, grâce à la cocainisation locale, et avec une sécurité plus grande, les expériences relatives aux effets de la compression ou de la ligature des artères coronaires. L'irritation faradique ventriculaire, qui tue le coeur normal, reste sans effet muisible su le coeur cocainé localement, quand les courants sont appliqués sur la zône cocainée; elle produit seulement une tétanisation incomplète et passagère du myocarde. D'accord avec les prévisions résultant des faits qui préçèdent, les poisons tétanisants du coeur perdent, en partie, leur influence, à la suite des applications locales de cocaine sur le coeur, ou de l'imprégnation du myocarde par la voie capillaire, après l'injection veineuse de doses suffisantes.''[20]

In 1931, Hermann and Jourdan[21] showed also that cocaine protects the dog's heart to a certain extent against certain causes of ventricular fibrillation. Mautz[18] and Beck and Mautz[22] also recommended the use of procain to reduce the cardiac irritability as a preventive measure against ventricular fibrillation and as an adjuvant in cardiac resuscitation. Their method, not different from the one used by previous workers, consists in surface application of procain solution or injection of the drug in the blood stream. Their results confirm the findings of François-Franck and many others.

To reduce cardiac irritability impeding resuscitation from ventricular fibrillation, we did not find that surface application of procain solution was a method giving as uniform and rapid results as the injection of the drug in the blood stream. It seems also that the injection through the auricles is a better method than the injection through the veins or the ventricles. In principle, it is believed that intraventricular injections should be avoided systematically in circulatory arrests because they may add complicating factors by the ventricular stimulation they provoke. One to 2 c.c. of a 2 per cent solution of procain are injected slowly through the left or right auricle. Very often, it will be found that approximately 0.50 c.c. is sufficient to stop paroxysmal ventricular tachycardia. The ideal dose in a given case can be determined by observing carefully the modifications of the cardiac rhythm. One should avoid doses causing considerable loss of tone and excitability of the heart. Indeed, if the organ is too much depressed, resuscitation will become difficult and will necessitate the use of other drugs to counteract the action of procain.

Here are two examples of the value of procain when cardiac hyperirritability prevents cardiac resuscitation.

Dog No. 237-45: This dog had a ligation of the ramus descendens of the left coronary artery at 2 cm. from the aortic origin of the left coronary artery. Ventricular fibrillation was initiated by clamping and declamping the aorta. In spite of massage, intra-auricular injection of epinephrin and electrical shocks, it was impossible to revive the heart definitely. After each electrical shock, the heart was defibrillated easily but immediately paroxysmal ventricular tachycardia developed followed by fibrillation. At the sixth defibrillation, while the heart was showing again paroxysmal ventricular tachycardia and before recurrence of fibrillation, one cubic centimeter of procain 2 per 100 was injected slowly in the left auricular appendage. Immediately the cardiac contractions slowed down, and without the help of any other procedure or medication, the heart was definitely resuscitated in spite of a ligature being left on an important coronary branch (Fig. 2.)

Dog No. 240-45: This dog had a ligation of the circumflex artery at 2 cm. from the origin of the left coronary artery. Eleven minutes and forty seconds later, asphyxia was produced by shutting off the air supply. Four minutes and thirty seconds after the beginning of asphyxia, the blood pressure was not recordable. At five minutes after the beginning of asphyxia, the air supply was resumed. It did not help the heart condition. The latter remained at standstill. Cardiac massage and epinephrin injection started ventricular fibrillation. The myocardium was very dilated and remained so in spite of prolonged massage. In certain instances, cardiac massage alone does not suffice to restore to the heart a lost cardiac tone. Barium chloride (1 c.c. of a 0.5 per cent solution) was injected into the left auricular appendage. The heart recovered its tone. Repeatedly thereafter electrical shocks defibrillated the myocardium. But each time, paroxysmal ventricular tachycardia occurred followed by fibrillation. One cubic centimeter of procain 2 per cent was injected into the left auricular appendage while the heart was fibrillating. It did not stop fibrillation, but one electrical shock applied immediately after the injection sufficed to defibrillate and resuscitate the heart definitely in spite of the ligature over the circumflex artery.

Wiggers and Wégria[23] concluded from their studies that procain raises the resistance of the ventricles to fibrillation but does not prevent its occurrence. They found no instance in which the heart became entirely refractory to fibrillation. Because they have witnessed instances in which repeated trials failed to defibrillate the ventricles, and, when they considered revival hopeless, still another shock was effective, they believe that the results of Beck and Mautz with procain may have been fortuitous.

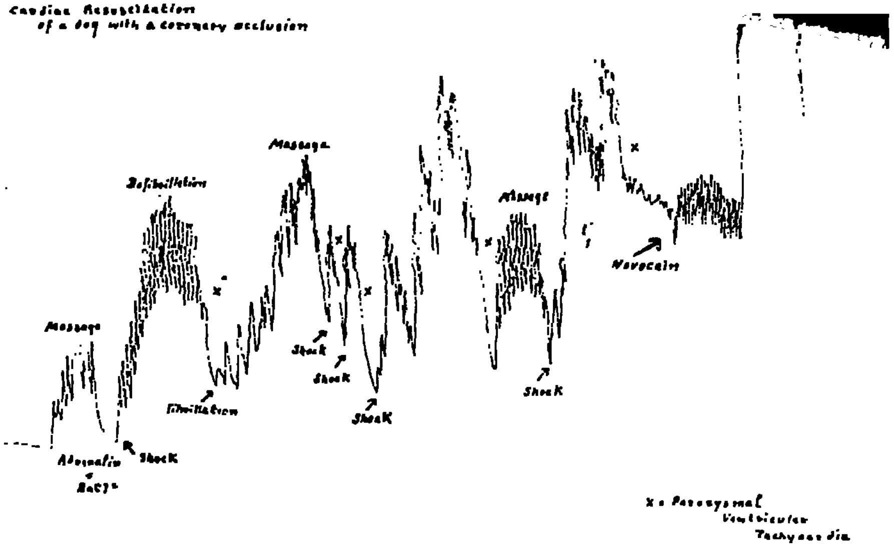

Fig. 2.—Blood pressure record illustrating the value of procain when cardiac hyperirritability prevents cardiac resuscitation. This dog had a coronary occlusion. Ventricular fibrillation was initiated by clamping and declamping the aorta. The heart was defibrillated each time that electrical shocks were applied to the myocardium; but each time, paroxysmal ventricular tachycardia (x on the tracing) developed rapidly, leading to ventricular fibrillation. Finally, while the heart was showing an extremely rapid ventricular rhythm again, and before the reappearance of fibrillation, one cubic centimeter of a 2 per cent solution of procain was injected into the left auricular appendage. Immediately the cardiac rhythm improved, the blood pressure came up, and the heart was definitely resuscitated.

That procain does not prevent fibrillation in every case is the conclusion reached by most of the investigators. No one expected that the drug could prevent the arrhythmia systematically. It is true also that it happens sometimes that after repeated failures while attempting to defibrillate the heart, a final electrical shock will prove effective. Yet, it is also true that such a final good result is not always obtained if one does not find and take care of the condition which impedes resuscitation. The fact that one electrical shock suffices in many cases to revive the heart after administration of procain when repeated shocks are without effect is so striking that it is hard to believe that such a result is purely fortuitous.

2. *Absence of Cardiac Tone.*—Sometimes, in spite of cardiac massage and intra-auricular epinephrin injection, the myocardium does not seem to recuperate

any tone. It remains dilated and flaccid. In such a condition, resuscitation is frequently impossible either from cardiac standstill or ventricular fibrillation. Prolonged massage is apt to cause bruises of the heart, endocardial ecchymoses which secondarily may seriously impair the cardiac conductivity. Experience showed us that barium chloride combined with epinephrin can help substantially to restore cardiac tone.

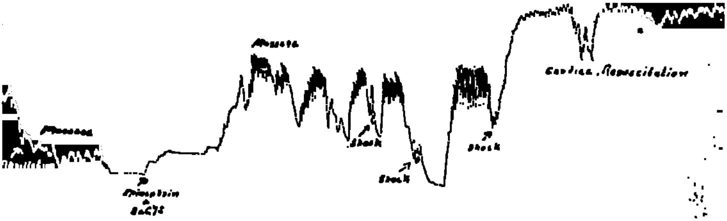

Fig. 3.—Blood pressure record illustrating the value of barium chloride when absence of cardiac tone prevents cardiac resuscitation. This dog had a coronary occlusion. The right coronary artery was tied at its origin. Sixty-two minutes later ventricular fibrillation developed. The heart had no tone. Massage did not help to restore it, as it frequently does. Epinephrin and barium chloride (2 c.c. respectively) were injected into the left auricular appendage. This time, cardiac massage increased the blood pressure, and after the third electrical shock, the heart was definitely resuscitated.

Bancroft and Dixon[24] found that barium chloride increases the force of the heartbeat, increases cardiac tone, and, in large doses, causes death in systole. Rothberger and Winterberg[25] state that it increases the irritability of the heart. After the administration of 5-10 mg. of barium chloride intravenously, the heart could no longer be brought to a standstill by a simultaneous stimulation of the sympathetic and vagus nerves, which they say is the case in the normal heart. Von Egmond[26] demonstrated that the ability of the ventricles to initiate rhythms was distinctly increased by the drug. Wertheimer and Boulet[27] state that it not only increases the cardiac excitability but that it can also help to prevent ventricular fibrillation when used in appropriate dosage. Tournade and Marchand,[28] Tournade and Giraud,[29] and Mathieu[30] also studied the action of barium chloride on cardiac irritability.

In our experiments, a solution of 0.5 per cent was used. It should be a fresh solution. Approximately 1 to 2 c.c. are injected through the left or right auricular appendage. One has to be cautious in the use of the drug. Overdoses can reduce considerably the amplitude of the systoles and lead to fatal results.

Here is one example showing the value of $BaCl_2$ to restore myocardial tone.

Dog No. 130-45: This animal had a ligation of the right coronary artery at its origin. Thirty minutes later, the circumflex branch of the left coronary artery was tied at its origin. Thirty-two minutes after that last coronary occlusion, ventricular fibrillation developed spontaneously. The blood pressure at that time was very low, and the heart was dilated and flaccid. The extensive deprivation of blood to the heart caused by both these ligations created the impression that resuscitation would be impossible. Cardiac massage did not improve the complete relaxation of the myocardium. Epinephrin and barium chloride, 2 c.c. of each, were injected separately into the left auricular appendage. The heart was massaged again. This time it recovered its tone. After the third electrical shock, the heart was resuscitated in spite of the fact that both ligatures were still in place. (Figs. 3 and 4.)

In many other experiments, we had similar good results.

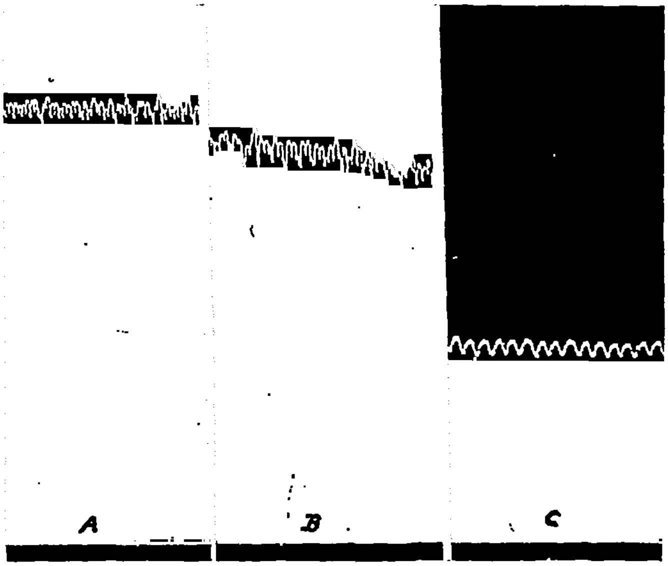

Fig. 4.—Continuation of the last blood pressure record. *A*, about three minutes after resuscitation. *B*, seventy minutes after the occlusion of the right coronary artery, and approximately seven minutes after resuscitation. *C*, twenty-four minutes after resuscitation.

INCREASE OF THE MYOCARDIAL TEMPERATURE

In certain difficult cases, it happens that one has to apply repeated electrical shocks to the heart in hopes that the last one may stop ventricular fibrillation. The repetition of these shocks, especially when administered in more or less rapid succession, will frequently increase the temperature of the myocardium quite remarkably. When such a condition exists, we find that it becomes practically impossible to defibrillate and resuscitate the heart. Cooling the organ with cold saline poured over the surface of the myocardium will in most instances allow restoration of the cardiac activity by the other means generally used. The effect is really striking.

Dog No. 247-45: This dog had a ligation of the ramus descendens at 2 cm. from the origin of the left coronary artery. Twenty minutes later, fibrillation developed. In spite of massage, the heart remained quite dilated. Epinephrin and barium chloride were injected into the left auricular appendage. The heart recuperated its tone. Yet electrical shocks applied five times in succession were without result. By that time, the temperature of the myocardium was quite elevated, and the heart was at complete standstill, undoubtedly the result of the high temperature. Sterile ice cubes were placed around the myocardium for a short while, and then removed. The heart was massaged again, and one cubic centimeter of epinephrin 1:1,000 was injected into the left auricular appendage. One electrical shock sufficed to resuscitate the organ definitely (Figs. 5 and 6).

PERIPHERAL CIRCULATORY FAILURE

Peripheral circulatory failure may develop during attempts to revive the heart either from cardiac standstill or ventricular fibrillation. It generally happens after long, unsuccessful trials to resuscitate the organ. Coordinated beats have been restored, and they seem to have sufficient strength to propel blood. Yet, surprisingly, the blood pressure remains at zero or at a very low level. Even cardiac massage, in those instances, does not seem to elevate it. To correct this condition, intracardiac infusions of Ringer or dextrose solutions and blood were tried. Blood was found, as it was expected, the best agent. Approximately 500 c.c. of blood are injected rapidly into the cardiac cavities through the left or right auricular appendage while massaging the heart. In many cases which seemed hopeless, such a procedure allowed resuscitation. When cardiac massage, which previously did not help in re-establishing suitable blood pressure, was begun soon after the beginning of the infusion, satisfactory elevation of the pressure resulted and continued when the heart was left working by itself.

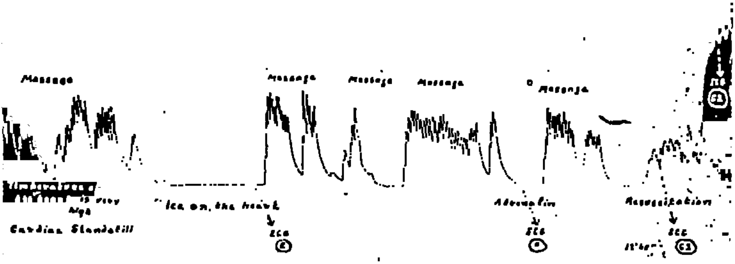

Fig. 5.—Blood pressure record, illustrating the value of cold saline or sterile ice cubes when increased myocardial temperature prevents cardiac resuscitation. This dog had a coronary occlusion. Ventricular fibrillation developed. In spite of massage, epinephrin, and repeated electrical shocks it was impossible to revive the heart. The myocardial temperature was very much increased. Ice cubes were applied on the surface of the heart. Later on, it was possible to revive the organ.

COMMENTS

From the very beginning of our work in cardiac experimental and clinical surgery, the importance of being familiar with the problems involved in cardiac failure and with the methods in use to revive a failing heart was fully recognized. The future of selective cardiac surgery depends to a very large extent on the development of technique subduing most of the serious operative risks involved in that type of surgery. The cardiac surgeon should be able to control and master the heart during operation. Otherwise, no security is possible; there is always present a feeling of impending disaster, and eventually dramatic fatalities can occur.

The skill necessary to operate with assurance and success on the myocardial structure can be gained only by considerable knowledge of the anatomy and physiology of the heart, accurate information and experience of many useful drugs, and, useless to say, extensive experimental surgery.

One must not be deceived by audacities crowned with success in traumatic cardiac surgery taking care of wounds representing a matter of imminent death. Cutler[31] states that surgical therapy in the general field of trauma needs no particular defense. It is the only treatment possible. Audacities and lack of foresight should not be allowed in selective surgery of the heart.

In the course of a cardiac operation, the essential accidents which must be prevented, or at least controlled, are: (1) hemorrhage, (2) serious circulatory disturbances due to unwise manipulations, (3) reflexes of various nature, (4) cardiac standstill, and (5) ventricular fibrillation. All these factors can cause immediate or rapid death.

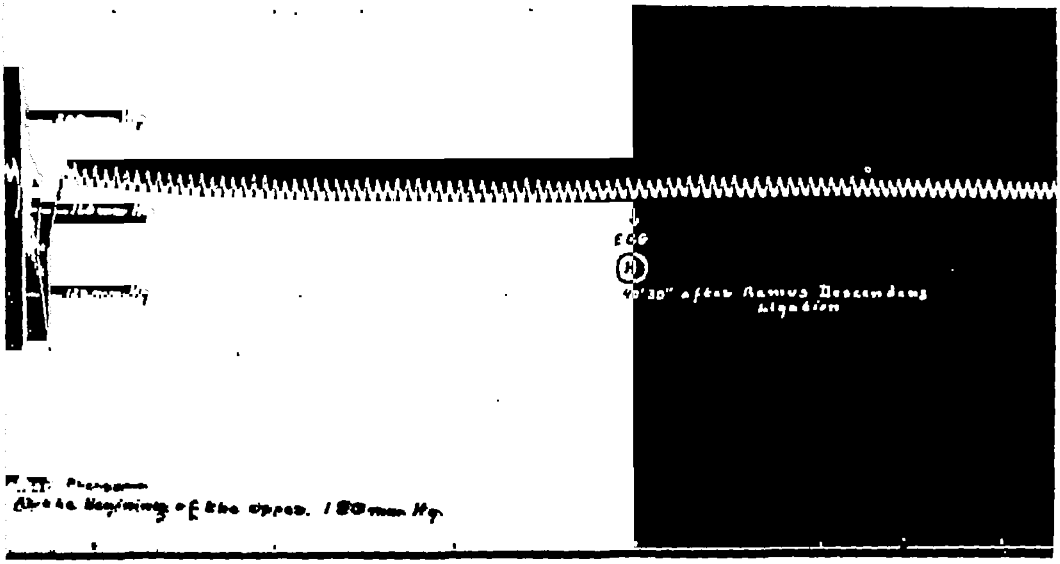

Fig. 6.—Continuation of the last blood pressure record. Approximately forty minutes after ligation of the ramus descendens, the blood pressure is exactly at the same level recorded at the beginning of the operation, that is, 180 mm. Hg.

Extremely valuable studies on the control of bleeding and reflexes have been published by physiologists interested in experimental cardiac work and by surgeons interested in both experimental and cardiac surgery. During the last two world wars, considerable experience has been acquired in the management of bleeding, reflexes, and avoidance of deleterious manipulations of the heart while treating cardiac wounds or extracting foreign bodies. It seems that now these three important factors of serious complications do not offer the same seriousness as in the past, and that they can either be avoided or taken care of with an increasing margin of safety. We have performed over twenty-five operations on the human heart for nontraumatic conditions. In no instance did bleeding, manipulations, or reflexes complicate the intervention or cause death of patients.

One cannot have the same assurance and optimism concerning the management of ventricular stoppage and ventricular fibrillation, especially when one keeps in mind that most cases coming for elective cardiac operation have a diseased heart or congenital malformation which often renders the organ a very fragile structure. Ventricular stoppage is not as easily controlled in such cases as in otherwise healthy young persons or rugged soldiers. Ventricular fibrillation is not as easily subdued as in dogs with healthy hearts in which ventricular fibrillation induced by electrical shocks is successfully treated by counter-shocks.

In our experiments on resuscitation, animals with healthy hearts were intentionally discarded in investigating the possibilities to revive the heart from ventricular fibrillation. In the experiments where cardiac standstill was induced, obviously the heart had to be damaged in advance. Therefore, it is believed that the conditions of experimentation were as near as possible to those found in clinical pathologic conditions.

Methods of resuscitation already described were used, as well as others which apparently are new. Our results confirm the results of previous experimenters on the value of cardiac massage, epinephrin, and electrical shocks. They also confirm their conclusions that members of the cocaine group can aid materially in reducing cardiac irritability and, therefore, in helping to prevent fibrillation. The use of barium chloride to restore cardiac tone seems something new in cardiac resuscitation attempts. The same is apparently true about cooling the myocardium. Intracardiac infusions of various fluids and blood have been tried before. Recently, and after the present work had been terminated, intracardiac blood infusions have been reported as valuable in cardiac resuscitation.

We believe that we have enough experimental evidence by now to show that with adequate equipment and a well-planned technique a higher percentage of good results can be obtained.

Every operating room should be equipped with a good automatic respirator, an electrocardiograph, a defibrillator, and thee ssential drugs required.

Aerating the lungs properly is essential. Manipulation of the thorax is recognized as essentially unworthy for adequate ventilation. Rhythmic compression of the rebreathing bag of the anesthetic machine is not a method of choice to establish and maintain artificial respiration. Aeration of the lungs should be carried out by an automatic respirator that will guarantee proper transport of oxygen at a regular rate, known volume and pressure, and for any extensive period of time. In some cases, natural and normal respiratory functions take a long interval before reappearing.

We believe that in the near future the electricardiograph will have a definite place in the operating room. It is indispensable during operation on the heart for various reasons. Electrocardiographic information is also essential during emergencies such as apparent death, especially when that happens while the chest is closed and, therefore, the cardiac behavior is unseen and cannot be estimated properly by the operator. The differential diagnosis between cardiac standstill and ventricular fibrillation can be made readily. A coordinated rhythm may be followed by incoordination during attempts to revive the heart from ventricular stoppage. The electrocardiogram will tell that immediately. This information is of the greatest assistance in guiding for defibrillation then as exists at the present time, this patient's life might have been saved. The patient had a compression scar on the heart. The scar had been cut through down to the heart muscle and was being rolled on the finger away from the heart, using sharp dissection. In doing this the descending ramus of the left coronary artery apparently was occluded, which produced ventricular fibrillation. Massage and adrenalin were of no value in restoration of the normal rhythm. The methods used by the physiologists at that time for defibrillation of the heart were of no practical value. They used potassium salts on the heart to get the heart out of fibrillary twitchings. This produced a large flabby heart. Calcium salts and adrenalin were then used to restore tone, without success. Then the method of shocking the heart, which was first described in 1899, was reintroduced. This method was not successful in our hands. The ventricles went back into fibrillation after the shock was applied. To this method we added the use of novocain which could prevent recurrence of fibrillation. This method was successful. This method of defibrillation can be applied to all those conditions that are of a reversible nature. These are the sudden deaths in the operating room from anesthesia, from drugs, from manipulation of thoracic viscera, etc.

What the surgeon usually does when this condition occurs is to give the patient artificial respiration and to inject adrenalin into the heart. Sometimes he will massage the heart by his hand. In a few minutes the brain sustains irreversible damage, the heart does not start, and nothing further can be done. And when the proper method of defibrillation is carried out, it has been my experience that anoxia has already killed the respiratory center before the oxygen system can be re-established. The breathing center must be kept alive if we are to be successful. This can be done by mechanical respiration plus massage of the heart. If the brain centers are kept alive then the heart can be successfully defibrillated.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—Would Dr. Fauteux tell something about the requirements of resuscitating agents with particular anesthetic substances? I understand that there is, for instance, a particular danger in producing ventricular fibrillation if one uses a combination of adrenalin with cyclopropane.

There was a very interesting article pertaining to the question in the *American Journal of Pharmacology and Experimental Therapeutics*, in 1939, by three or four authors, one of whom was Orth and another Leigh. They showed in that article that there was a particular danger of throwing the heart into ventricular fibrillation if adrenalin is injected in the course of cyclopropane anesthesia; however, neosynephrin was a much safer substance to use.

DR. OTTO C. BRANTIGAN, Baltimore, Md.—I would like to report a rather unusual cardiac resuscitation in a 24-year-old white girl.

Arrest of cardiac motion occurred from no demonstrable cause at the completion of the intrathoracic portion of a lobectomy of the upper lobe of the right lung. An intratracheal tube was in place and by this means the lungs were kept well inflated with oxygen throughout the period of resuscitation. Ordinary massage of the heart was first carried out with only twitching of the heart in response. It soon became evident that the more vigorous manipulation of the heart gave better results, since a few beats of the heart were observed when the hand was removed from the heart. Finally the hand was forced posterior to the heart and the other hand placed over the rather pliable sternum; thus, bimanual pressure and release was applied at the full strength of the operator. Each time the hand was removed for observation of the heart, a few beats would be evident, but arrest of its action would again occur. The operator was unable to carry on uninterruptedly, and accordingly was relieved by the first assistant. Thereafter pressure and release were carried out alternately by the surgeon and the first assistant.

After about twenty-five minutes it seemed ridiculous to continue, but each time the hand was removed, twitchings or a few beats of the heart were evident before complete cardiac arrest again occurred. At the end of thirty-five minutes of such vigorous manipulation, the heart rather suddenly began to beat spontaneously. The rate of beat rapidly increased to about 120 per minute and in a very few minutes the blood pressure was in the neighborhood of 80 mm. of mercury.

Closure of the chest wall was delayed for fifteen minutes in order to allow time for observation and more complete recovery of the heart. The operation was completed and the patient made an uneventful recovery. She did not develop mental changes nor cerebral manifestations of any kind.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—All the cases of pulmonary emboli which I have operated on, which number twenty-three, have had heart arrest for a time.

The type of resuscitation which we have used has been novocain in the pericardium itself, vigorous massage, taking the heart in the hand and squeezing it in about the same rhythm as the normal heartbeat, and adrenalin 1/1,000 intracardially. In about half of those cases we have had, we have been able to get the heart going again.

The ones who died, all died because of cerebral damage, caused by too long a period of anoxemia of the brain, not from the heart itself.

In dogs I have had the aorta and the pulmonary artery clamped together at the heart base with a clamp for as long as up to twenty-three minutes. Actually, during the time those vessels were clamped, we had the brain of that dog circulated from another dog lying beside it.

We used the same type of resuscitation and were able to get heart action again.

The heart in the clamped dog did not stop completely before from three to six minutes after the clamp was placed.

I have had dogs living and quite normal, about a year after this operation.

DR. MERCIER FAUTEUX, Boston, Mass.—Direct myocardial stimulation of any kind during cardiac standstill can provoke ventricular fibrillation as long as cardiac ischemia still associated with an abnormal degree of cardiac irritability exists. These two conditions, apparently essential for the initiation of ventricular fibrillation during cardiac standstill are, indeed, likely to appear in a variable time, depending mostly on the state of the myocardial tissues before cardiac arrest.

I agree with Dr. Beck that the old methods to revive the heart after ventricular fibrillation by administration of potassium and calcium are not practical.

About the use of members of the cocaine group to decrease cardiac irritability and prevent ventricular fibrillation, it is an old procedure known as valuable since 1884. It was advocated by François-Franck and used by many others in the past years.

I believe that any type of general anesthesia, chloroform, ether, as well as cyclopropane, can determine ventricular fibrillation when adrenalin is administered during the period of induction or at any time the patient is under light anesthesia. When anesthesia is deep, the drug is apparently less apt to initiate ventricular fibrillation. These facts have been emphasized by Levy and many other workers. The mechanism of fibrillation in such instances is apparently still obscure. Probably general anesthetics can have at a certain moment a depressive action on the conductive system and in such a condition cardiac stimulation provoked by adrenalin is sufficient to create heterotopic centers leading to fibrillation.

TWO UNUSUAL TUMORS OF THE STERNUM

THOMAS J. KINSELLA, M.D., S. MARX WHITE, M.D., AND R. W. KOUCKY, M.D.
MINNEAPOLIS, MINN.

TUMORS of the sternum are of rare occurrence. Even in the large clinics, the number of cases reported is very small in proportion to other types of tumors, with Crile, in 1936, reporting only sixteen from the complete files of the Cleveland Clinic, and Macey and Phalen, in 1943, only fourteen from the entire Mayo Clinic experience. The occurrence of two of the rarer types of sternal tumors in the practice of one physician within a single year is all the more phenomenal with such a background.

Tumor masses of the sternum may be primarily benign, malignant, metastatic, or inflammatory in their origin. Metastatic tumors occur most commonly, with primary malignant growths next, then the inflammatory masses, and last and rarest of all, the primary benign tumors. The task of attempting to compile all recorded cases is a very difficult one because of incomplete reports, inadequate descriptions, and confusing pathologic diagnoses. It has been aided very materially by the excellent collective reviews on the subject presented previously by Parham, Heuer, Hedblom, Crile, Sommer and Major, and others. The following tabulations of cases of the various types collected from the literature will give an adequate idea of the types and relative frequency of each variety of tumor.

METASTATIC TUMORS

Metastatic tumors are encountered more frequently than any other condition affecting the sternum, and constitute nearly 38 per cent of the reported cases. There must be very serious doubt, however, that anything approaching a complete recording of all metastatic lesions to the sternum appears in the literature. Carcinoma of the thyroid appears to be the most frequent primary tumor to give sternal metastases, with twenty-three such cases recorded, while carcinoma of the kidney or hypernephroma appears next, with twelve. Both of these tumors commonly give pulsating metastatic masses in the sternum (eleven of twenty-three carcinomas of the thyroid, and nine of twelve of hypernephroma). These are the only sternal tumors, either primary or secondary, in which expansile pulsations have been described. Nine cases of lymphoblastoma, and thirteen of carcinoma of the breast are recorded, with only scattered cases from other primary sites. Any portion of the sternum may be involved, though the upper segment is affected slightly more frequently (manubrium seventeen, sternal clavicular joint nine, entire sternum eight, and gladiolus ten times). As might be expected from the nature of the conditions presented, treatment has been rather unsatisfactory; excision operations

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Read (by title) at the Twenty-Sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30 and 31, 1946.

have not produced results to justify the extensive surgical procedure involved, and radiation therapy, except in the lymphoblastoma group, has not proved much better.

PRIMARY MALIGNANT TUMORS

Primary malignant tumors of the sternum appear second in frequency to the metastatic lesions, with fifty-two cases recorded. As one would expect, the majority of these tumors have been sarcoma of one type or another. A variety of types of sarcoma have been recorded. The cases have been almost equally divided between the two sexes, with eighteen reported in males and sixteen in females. The age distribution has varied from 12 to 69 years of age, with a rather even scattering over the various decades, four cases are recorded between 12 and 20, four cases from 21 to 30, eight cases from 31 to 40, eight cases from 41 to 50, six cases from 51 to 60, and one at the age of 69. The manubrium was involved in fourteen, the sternoclavicular joint in one, the gladiolus in thirteen, the whole sternum in two, and the xiphoid in one patient.

The records are incomplete in many of the cases. Twenty-six of the tumors were treated by resection of a more or less radical type, with twenty recoveries and five deaths from surgery. Eight of these patients who recovered from the surgery are recorded as showing recurrence sometime later, with no late reports in nine patients. From the figures available, it would appear that the results presented would seem to justify radical resection on a short-term basis, but that late results cannot be evaluated from the data available. Curettage or local excision would appear to offer little in a curative way. Two patients treated by Coley's toxins were reported as improving, but no late results were reported. From the literature reviewed, there would appear to be insufficient data to evaluate the results of radiation therapy.

Among the other cases of malignant tumor of the sternum reported in the literature, there is one patient with myeloid sarcoma reported by König in 1882, and a myelosarcoma by Angelescu and Stoia in 1932, a "myeloid tumor" which could have been a carcinoma of the kidney, by Malloizel in 1904, an endothelioma, by Macey and Phalen in 1943, a vascular sarcoma or aneurysm unconfirmed by operation or biopsy, by Zinninger in 1930 (a pulsating tumor), two cases of myeloblastoma, by Macey and Phalen in 1943, and "myeloma," by Turner in 1915, unconfirmed by biopsy operation or blood studies and treated by radium with recovery and return to work. There has been but one case of solitary plasma cell myeloma reported in 1938 by Scheinker with involvement of the gladiolus. No treatment was given, the patient dying soon from a Landry's paralysis.

BENIGN TUMORS OF THE STERNUM

The commonest benign tumor of the sternum reported in the literature is chondroma or enchondroma, of which eleven cases are recorded. Four of these have been described as chondroma, four as enchondroma, one as a vascular enchondroma, and one as an osteochondroma. An additional case reported by Heuer as chondroma has been listed with the sarcomas because of

TABLE I. METASTATIC TUMORS

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
1	1862	Legroux	M	74	Gladiolus	Nuclear carcinoma	No operation	Died 1 year: tuberculosis also
2	1868	Behier	F	45	Gladiolus	Carcinoma	None	Died: also had syphilis
3	1868	Ollivier	F	53	Manubrium	Carcinoma of thyroid (?)	None	Died: pulsating
4	1890	Damourette and Claisse	F	56	Manubrium and sternum	Carcinoma of thyroid	None	Died: pulsating
5	1891	Mynter	F	22	Gladiolus	Melanosarcoma	Resection of gladiolus	Recovered: died one year later, metastases
6	1893	Graves	F	44	Gladiolus	Carcinoma of breast	Resection of gladiolus	Recovered: no late result
7	1893	Von Eiselberg	M	72	?	Carcinoma of thyroid	Resection	Died
8	1897	Keen	F	44	Manubrium and gladiolus	Carcinoma of breast	Partial resection of manubrium and gladiolus	Recovered: died 5 months later
9	1897	Carle	F	50	?	Carcinoma of thyroid	—	Died 14 days later: pulsating
10	1901	Oderfeld and Steinhaus	M	58	Manubriosternal joint	Carcinoma of thyroid	—	—
11	1901	Helbing	F	51	Manubrium	Carcinoma of thyroid	None	Died: pulsating
12	1902	Morestin	F	?	Manubrium	Sarcoma of breast	Resection of manubrium	—
13	1902	Yoder	F	51	Manubriosternal and gladiolus	Carcinoma of uterus	—	—
14	1904	Pautrier	M	68	Gladiolus	Carcinoma of testicle?	—	General metastases
15	1904	Halbron	F	68	Manubriosternal joint	Carcinoma of thyroid	—	Died 3 years: pulsating
16	1904	Malloizel	M	21	Sternoclavicular	Carcinoma of kidney?	None	Died: involvement of lung, mediastinum, and lymph nodes
17	1905	Rixford	F	45	Gladiolus	Carcinoma of breast	Resection to cartilage and edge of sternum	Recovered: died three months later
18	----	Rixford	F	53	Manubrium: gladiolus	Carcinoma of breast	Two previous minor resections: resection of sternum above 5th rib	Recovered: no late report

19	----	Rixford	M	70	Sternoclavicular joint	Endothelioma. Sarcoma of clavicle	Resection clavicle and upper sternum	?
20	1906	Doreum	F	56	Sternum, clavicle	Carcinoma of thyroid	—	Died
21	1907	Langhans	M	38	Sternum	Carcinoma of thyroid	—	Died
22	1908	Eschner	--	--	—	Hypernephroma	—	—
23	1909	Porelle	F	46	Inner clavicle and *manubrium*	Carcinoma of thyroid	Extirpation	Died: cerebral and spinal metastases
24	1909	Boinet	M	53	Whole sternum	Hypernephroma	—	Died pulsating
25	1912	Lanphear	M	61	Manubrium	Carcinoma of breast	Two-stage resection manubrium and clavicle	Recovered: no late result
26	1921	MacLeod and Jacobs	M	54	Manubrium and gladiolus	Hypernephroma	Biopsy	Died two months
27	----	MacLeod and Jacobs	M	69	?	Hypernephroma	—	Died 6 weeks: pulsating
28	1924	Louste-Thibaut, Valiere-Vialeix	—	—	—	Hodgkins	—	—
29		Emmerich	F	68	?	Carcinoma of thyroid	—	—
30	1926	Cabot-Dresser	M	40	Manubrium	Lymphoblastoma	X-ray treatment	Alive 5 years
31	1926	Dresser	M	35	Manubrium	Lymphoblastoma	X-ray treatment	Recovered, 10 months
32		Dresser	F	10	Gladiolus	Lymphoblastoma	X-ray treatment	No late report
33		Dresser	M	40	?	Lymphoblastoma	X-ray treatment	Died 14 months
34	1926	Maingot	F	73	Manubrium	Carcinoma of thyroid	Biopsy	No result
35	1928	Biancheri	--	--	—	Carcinoma of thyroid	—	—
36	1930	Cabot	M	59	Gladiolus	Hypernephroma	Biopsy and x-ray treatment	Pulsating
37	1931	Low Beer	--	--	—	Carcinoma of thyroid	—	—
38	----	Low Beer	--	--	—	Carcinoma of thyroid	—	—
39	----	Low Beer	—	--	—	Carcinoma of breast	—	—
40	1934	Dinsmore and Hicken	F	42	Sternoclavicular	Carcinoma of thyroid		Living 8 years
41	1934	Singleton	F	55	Manubrium	Carcinoma of thyroid	Resection of manubrium	Pulsating, bruit
42	1936	Crile	M	53	Gladiolus	Hypernephroma	Local enucleation	Died, accidental 7 weeks: pulsating bruit
43	----	Dinsmore, Hicken and Crile	F	63	Manubrium	Carcinoma of thyroid		Died 4 years: pulsating

TABLE I—(CONT'D)

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
44	----	Dinsmore, Hicken and Crile	M	57	?	Carcinoma of thyroid		Pulsating
45	----	Crile	M	63	Manubrium	Carcinoma of thyroid		Pulsating
46	----	Dinsmore, Hicken and Crile	M	68	?	Carcinoma of thyroid		Pulsating, bruit
47-50		Crile	4 cases		--	Carcinoma of breast	—	—
51	1937	Fonts-Abreu	F	39	?	Hodgkins	X-ray treatment	O.K. 16 months
52	1937	Roth and Davidson	F	55	Entire sternum	Hypernephroma		Pulsating, died
53	1939	Paraf, Fischgold, Abaza, Lewi, Behrend	M	26	Manubrium	Hodgkins	Biopsy x-ray	—
54	1939				Manubrium	Hodgkins	Resection x-ray treatment	O.K. 1½ years
55	1940	Lewis	M	48	Manubrium	Hypernephroma		Pulsating
56		Lewis	M	58	Sternoclavicular, manubrium	Hypernephroma		Pulsating
57	1942	Horsley	M	74	Manubrium	Carcinoma of kidney?	Curetted	Pulsating
58	1942	Sommer and Major	F	28	Gladiolus	Hodgkins	Partial excision	Died 2 years
59	1943	Molle	M	64	Manubrium	Carcinoma of thyroid	—	Pulsating: died 8½ years
60	1943	Macey and Phalen	--	--	—	Carcinoma of breast	—	—
61	----	Macey and Phalen	--	--	—	Carcinoma of thyroid	—	—
62	----	Macey and Phalen	--	--	—	Carcinoma of stomach	—	—
63	----	Macey and Phalen	--	--	—	Carcinoma of rectum	—	—
64	----	Macey and Phalen	--	--	—	Hypernephroma	—	—
65	----	Macey and Phalen	--	--	—	Carcinoma of bronchus?	—	—
66	----	Macey and Phalen	--	--	—	Carcinoma of bronchus?	—	—
67	1946	Kinsella	F	44	Manubrium	Carcinoma of breast	—	—

the metastases which appeared within the year. Another patient reported by Dudon in 1890 as having enchondroma had had a previous operation twenty-seven months before, was then treated by resection of the manubrium with a portion of the gladiolus with recovery followed by local recurrence, which may indicate that the tumor was of low-grade malignancy. Five were treated by more or less radical resection with recovery, and two by local excision, one dying of pyemia in five days, the other recovering. Two cases were reported by Sommer and Major in 1942, one treated by resection was reported free of trouble seven and one-half years later, another treated by local removal was likewise well four years later. No late results are available in the other cases.

A series of six tumors was reported by Bardenheuer in 1885 as "fibroid tumors" of the manubrium, treated by radical resection with four recoveries and two deaths. No pathologic diagnoses were given and no late results reported. Other cases recorded include two cases of cyst of the sternum, one listed as a congenital serous cyst and one as a dermoid cyst, and a case of Von Recklinghausen's disease involving the sternum reported by Gougerot and Meyer-Heine in 1935. From these reports it would appear that benign tumors of the sternum may be treated by resection with satisfactory results without undue danger. As a considerable number of the reported cases of resection were done years ago before modern thoracic surgery developed, the results must be considered excellent, and even better may be expectd under modern handling.

INFLAMMATORY MASSES

There are thirty-three instances of inflammatory masses involving the sternum which we have collected from an incomplete review of the literature. These have been derived from a number of causes, including acute and chronic osteomyelitis, tuberculous osteomyelitis, gumma, acute abscess, and, more recently, eosinophilic granuloma. In addition to this, echinococcic involvement has been reported with increasing frequency (thirteen cases). A report by Bauer in 1912 mentioned four cases of sternal involvement among 243 cases of bone involvement with echinococcus cyst. Pasquale, in 1930, reported nine cases among 406 additional bone cases of echinococcus disease. Sinberg, in 1936, reported a patient treated by drainage with recovery. Inflammatory lesions of the sternum are not likely to be confused with tumor if a careful study of the condition is made. Drainage had been carried out in a number of instances, with resection in others, even in tuberculous osteomyelitis.

CASE 1.—A. C. K., aged 33, was first seen on April 18, 1945, complaining of a growing lump on his breastbone. His past history was entirely negative except for a tonsillectomy in 1941 and a cholecystectomy in November of 1943. He had been perfectly well until late August, 1944, when, while pitching in a game of softball, he was struck over the sternum by a line drive. He stated that the blow hurt at the time but that the skin was not broken, there was no disability, and he continued pitching that game and pitched another later the same afternoon. He then forgot about the incident until sometime later, perhaps in late October, when he first noticed a lump appearing over the body of the sternum. This increased slowly in size until January, 1945, following which it seemed to grow more rapidly. At times the lump was somewhat tender, but there was no spontaneous pain except occasion-

TABLE II. PRIMARY MALIGNANT TUMORS

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
1	1873	Weinlechner	M	58	?	Colloid sarcoma	Local excision	Died 5 days, pyemia
2	1878	Holden	F	52	Gladiolus	Calcified sarcoma	Local removal	No follow-up
3	1882	König	F	36	Gladiolus	Myeloid sarcoma	Resection gladiolus, part of manubrium, and adjacent ribs	Recovered: Died 2 years, recurrence
4	1884	Pfeifer	M	45	Gladiolus	Sarcoma	Resection of gladiolus, and ribs 2, 4 A & L	Died six days, metastases
5	1885	Bardenheuer	M	--	Clavicle and manubrium	Sarcoma	Resection manubrium, 2/3 clavicle, and ribs 1 and 2	Recovered; no follow-up
6	1887	Jaenel	F	12	Manubrium	Sarcoma	Resection of manubrium	Recovered; ok 1 year
7	1890	Mazzoni	M	55	Gladiolus	Myxosarcoma	Resection gladiolus and ribs, 2 and 4	Died
8	1893	Welch-Unger	F	33	Manubrium	Medullary sarcoma	Drained and curetted	Died ten months
9	1893	Coupland and Gould						
10	1894	König	M	59	?	Sarcoma	Resection sternum and ribs 2 and 5	Died
11	1894	Park	M	26	Whole sternum	Chondrosarcoma	Explored, inoperable	
12	1895	Doyen	M	37	Manubrium	Sarcoma	Resection manubrium and clavicles	Recovered; no late report
13	1897	Keen	F	28	Manubrium	Sarcoma	Resection manubrium, 1/3 clavicle, and rt. 1st rib	Recovered: o. k. 15 months
14	1897	Rosa	F	16	Manubrium	Sarcoma	Inoperable	Died 3 months
15	1898	Thurston	F	61	Gladiolus	Periosteal sarcoma	Coley's fluid	Smaller, no late results
16	1901	Amburger	M	47	Manubrium	Round cell sarcoma	Resection manubrium and rt. ribs 1 and 2	Died 3 days
17	—	Amburger	M	37	Gladiolus	Round cell sarcoma	Resection gladiolus and three ribs	Recovered: died of metastases
18	—	Amburger	F	22	Manubrium	Spindle-cell sarcoma	Resection of manubrium and ribs 1 and 2	Recovered: o. k. 2 years
19	—	Amburger	M	40	Manubrium	Fibromyxosarcoma	Resection of manubrium and rt. ribs 2 and 3	Recovered: died 1 year, recurrence

20	1902	Griffiths	M	25	Gladiolus	Round cell sarcoma	Incomplete resection gladiolus	Recovered: died 4 months recurrence
21	1902	Christ						
22	1903	Cavillon and Jaboulay	M	—	Manubrium	Osteosarcoma	Resection	Recovered: no late result
23	1904	Mayer	M	38	Manubrium	Sarcoma	Resection of sternum and rt. ribs 1, 2, and 3	Died, empyema 9 days
24	1904	Malloizel	M	21	Manubrium	Myeloid tumor, could be carcinoma of kidney	--	Died, tumor lungs, mediastinum, and axillary nodes, rt., kidney and sternum
25	1905	Carson	--	—	Gladiolus	Osteosarcoma	Resection gladiolus and ribs 3 and 4	Recovered, flapping chest: No late report
26	1908	Coley	F	38	Manubrium	Spindle cell	Biopsy, Coley toxins	Almost gone 7 months: no late results
27	1909	Isaacs	F	46	Gladiolus	Spindle cell	Resection of gladiolus	Recovered, recurrence and metastases 1 month
28	1909	Tixier and Gangol	F	39	Manubrium	Malignant enchondroma	Resection of manubrium, inner clavicle, and rt. and lt. ribs 1 and 2	Recovered: wore corset, no late report
29	1914	Le Jars	M	40	Xiphoid	Sarcoma	Resection of xiphoid	Recovered: no result stated
30	1915	Turner	M	32	--	No biopsy, myeloma	Radium, 13,200 mg. hr.	Tumor bone 1 month, recovered and returned to work
31	1910-1920	Aimes and Antonin	F	15	Manubrium	Globlet cell sarcoma	Biopsy, inoperable	
32	1930	Zinninger	M	47	Gladiolus	Vascular sarcoma, aneurysm	No operation, no biopsy radium	No improvement, pulsated
33	1931	Goldman	M	49	Gladiolus	Chondrosarcoma	Resection gladiolus and ½ manubrium	Recovered: recurrence 3 years
34	1931	Low-Beer	--	--	--			
35	1932	Vitale	M	19	?	Small round cell sarcoma	Biopsy only	
36	1932	Angelescu and Stoia	--	--	--	Myelosarcoma	--	
37	1932	Heuer	F	56	Gladiolus	Chondromyxosarcoma	Resection gladiolus	Recovered: metastases 1 year later

TABLE II—(CONT'D)

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
38	1933	Suwalski and Danielewski	--	--	Sternoclavicular	Mixed neoplasm	--	
39	1935	Roberg	F	39	Manubrium and gladiolus	Chondrosarcoma	2 resections and x-ray treatment	Recovered: ? recurrence 6 months
40-1	1936	Crile	--	--	Sarcoma	--	--	--
42-3	—	Crile	--	--	--	--	--	--
44	—	Crile	--	--	--	--	--	--
45	1936	Crile	--	--	--	Cavernous hemangioma	--	--
46	1940	Bradshaw and Chodoff	F	49	Manubrium	Chondrosarcoma	Resection manubrium and inner clavicle	Recovered o. k. 23 months
47	1941	Cauble	M	54	Gladiolus	Osteogenic sarcoma	Resection gladiolus, x-ray treatment	Recovered
48	1942	Sommer and Major	M	49	Gladiolus	Chondrosarcoma	Resection gladiolus	Recovered: no late report
49	1943	Macey and Phalen	--	--	--	Chondrosarcoma	--	--
50	—	Macey and Phalen	--	--	--	Chondrosarcoma	--	--
51	—	Macey and Phalen	--	--	--	Chondrosarcoma	--	--
52	—	Macey and Phalen	--	--	--	Chondrosarcoma	--	--
53	1943	Macey and Phalen	--	--	--	Myeloblastoma	--	No details
54	1943	Macey and Phalen	--	--	--	Endothelioma	--	--
55	1943	Macey and Phalen	--	--	--	Myeloblastoma	--	No details
56	1944	Woodward	M	--	--	Resected x-ray		No report
57	1946	Kinsella, White, and Koucky	M	60	Manubrium	Plasma cell myeloma	Resected	Recovered: o. k. 1 year

ally when coughing. There had been no other symptoms and his general health had been good. There has been a slow, steady gain in weight.

Physical examination revealed a well-developed, well-nourished adult white male, 6 foot 1 inch tall, weighing 170 lb. The temperature was normal, the pulse 70 and the blood pressure 122/70 mm. mercury. The thyroid showed a small, firm nodule in the right lobe, but no signs of hyperthyroidism. The chest and heart were negative except for the findings in the sternum which presented a raised, slightly irregular, nodular prominence of a bony consistency occupying the entire gladiolus, measuring 5 by 12 cm., and raised 1.5 cm. above the normal sternal level. The overlying skin showed no change and was not adherent. There was definitely no pulsation present, and no bruit or thrill. The abdominal findings were negative except for a right upper rectus scar.

The hemoblobin was 15.5 Gm. (92 per cent), erythrocyte count 4,650,000, leucocyte count 7,100, polymorphonuclear leucocytes 63 per cent, lymphocytes 19 per cent, monocytes 9 per cent, eosinophiles 8 per cent, and basophiles 1 per cent. The sedimentation rate was 7 mm. The blood complement fixation test for syphilis was negative. The blood phosphorus was 4.18 mg. per cent and the blood calcium was 10.53 mg. per cent. The coagulation time was 8 minutes and 30 seconds, the bleeding time 3 minutes and 10 seconds. Blood grouping was group IV (O), Rh positive. The urine specimen showed a pH of 6.5, specific gravity 1.020, albumin and sugar negative, one epithelial cell and one leucocyte per high power field microscopically. Examination of the urine for Bence-Jones protein was negative. A twenty-four-hour urine specimen, 1,980 c.c., showed 18.06 mg. phosphorus per 100 c.c., total phosphorus 357.89 mg. per 24 hours, calcium 13.42 mg. per 100 c.c. and total output of calcium 265.72 mg. per 24 hours.

X-ray examination of the chest on May 3, 1945, revealed the diaphragm, heart, and mediastinal shadows as normal and the lung fields clear. X-ray films of the ribs, skull, and both wrists showed no evidence of destructive bone lesion. X-ray films of the sternum made in oblique and lateral positions and planigrams show a rather marked irregularity in outline of the body of the sternum, there being loss of the trabecular pattern which appears to be replaced by a homogeneous type of density which is rather markedly reduced from the normal. The findings, therefore, suggest some type of tumor of the body of the sternum, which has largely infiltrated the entire bony structure, has produced rather marked decrease in the density as well as the trabecular pattern, and has produced an irregularity in outline, especially along the medial margins. The findings suggest some type of cystic tumor which is apparently of a destructive nature.

A tentative diagnosis of primary benign giant cell tumor of the sternum was made. Several diagnostic possibilities were considered before this diagnosis was reached. Multiple myeloma was excluded on the basis of the patient's age (33), the absence of involvement of the ribs and skull, the character of the tumor itself with an intact bone shell and the bony septa extending through it, and the absence of Bence-Jones protein in the urine. The possibility of this lesion being a manifestation of osteitis fibrosa cystica of parathyroid origin was likewise considered, because of the palpable mass in the thyroid. This was ruled out as a probable diagnosis by the normal x-ray of the ribs on both sides, the normal blood calcium and phosphorus values, and the normal urinary calcium and phosphorus. A thorough search was made for other primary tumor sites, including the kidneys, without finding a primary tumor from which this could be metastatic. The possibility of a primary malignant tumor was likewise considered, but the x-ray picture of the intact bony shell and lack of evidence of local invasion were considered to favor a benign growth. Excision of the involved portion of the sternum, therefore, was recommended.

The question of the advisability of removal of a biopsy specimen was considered but rejected on the ground that a delay would be entailed, following the biopsy by the necessity for decalcification and preparation of the material for study, that it seemed inadvisable to break the apparently intact shell of the tumor mass, and because the same contemplated procedure would be carried out in spite of the biopsy report. The operation was, therefore, planned as a total excision of the involved portion of the sternum with adjacent cartilages, and because of the size of the tumor and the wide defect which would be left, reconstruction of a stable chest wall at conclusion of the operation was decided upon.

TABLE III. BENIGN TUMORS

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
1	1870	Turner	--	--	--	--	--	--
2	1873	Weinlechner	M	58	--	Chondroma (Also had sarcoma)	Local excision	Died, pyemia 5 days
3	1880	Bigelow	--	--	--	Enchondroma?	--	--
4	1880	Bigelow	--	--	--	Enchondroma?	--	--
5	1833	Lisfranc	M	44	Manubrium	?		No late report
6	1885	Bardenheuer	F	--	Manubrium	"Fibroid tumor"	Resection manubrium, 2/3 clavicle, and ribs 2 and 3	Recovered: no late report
7	--	Bardenheuer	--	--	Manubrium	?	Resection manubrium	Recovered: no details
8	--	Bardenheuer	--	--	Manubrium	?	Resection manubrium	Died
9	--	Bardenheuer	--	--	Manubrium	?	Resection manubrium	Died
10	--	Bardenheuer	F	--	Manubrium		Resection manubrium and part of gladiolus, inner clavicle	Recovered: no late report
11	--	Bardenheuer	--	--	Manubrium	?	Resection manubrium, part of gladiolus, inner clavicle, and right ribs 1 and 2	Recovered: no late report
12	1890	Dudon	F	28	Gladiolus	Enchondroma	Previous operation 27 months before: resection manubrium and part of gladiolus	Recovered: local recurrence
13	1893	Lanelongue	--	--	--	Congenital serous cyst		
14	1893	Coyne and Cannieu	--	--	?	Vascular enchondroma	Resected	No report
15	1904	Wiesmuller	--	--	--	Chrondroma	--	--
16	1904	Bird	--	--	--	Dermoid cyst	--	--
17	1911	Lusena	M	56	Gladiolus	Chondroma	Partial resection	Recovered
18	1913	Richardson	M	26	Manubrium	Enchondroma	Resection of manubrium	Recovered: no late results
19	1935	Gougerot and Meyer-Heine	M	46	?	Von Recklinghausen's disease	None	None
20	1936	Crile				Chondroma		
21	1942	Sommer and Major	M	34	Gladiolus	Chondroma	3 operations, resection	Recovered: o. k. 7½ years
22	--	Sommer and Major	M	26	Manubrium, Gladiolus	Osteochondroma	Local removal	Recovered: o. k. 4 years
23	1946	Kinsella-White and Koucky	M	33	Gladiolus	Benign giant cell tumor	Excision of gladiolus	Recovered: o. k. 1 year

TABLE IV. INFLAMMATORY

NO.	YEAR	AUTHOR	SEX	AGE	LOCATION	DIAGNOSIS	TREATMENT	RESULT
1	1860	Bizzoli	F	29	Manubrium and gladiolus	—	Subperiosteal resection, sternum	—
2	1870	Wilde	—	—	—	Caries of tarsus, metatarsals and body of sternum	Resection of sternum	—
3	1882	Kuster	M	30	Gladiolus	Gumma	Resection one-half gladiolus and ribs 3 and 4	Recovered, no late report
4	1882	Rich and Bowen	—	—	Gladiolus	Acute necrosis	—	Pulsating tumor
5	1885	LeFort	M	54	Gladiolus and sternoclavicular	Abscess	Recovered	Died three years
6	1886	Molliere	—	—	—	Abscess of breast and sternum	Resection	—
7	1889	Hewitt	—	—	—	Caries	Partial removal	—
8	1893	Yvert	—	—	—	Osteoarthritis	Resection	Subluxation
9	1894	Desguin	—	—	Manubrium	Tuberculous osteomyelitis	Resection	—
10	1896	Delore	—	—	Gladiolus	Tuberculous osteomyelitis	Resection	—
11	1903	Stone	—	—	—	Gumma	—	—
12	1904	Sencert	—	—	—	Cold abscess	Resection	Recovery
13	1912	Bauer	—	—	—	Echinococcus	—	Four cases in 243 bone cases
14	1921	Hedblom	—	—	—	Chronic inflammation	Resection	—
15	1930	Pasquale	—	—	—	Echinococcus	—	Nine cases of 406 bone cases
16	1931	Low Beer	—	—	—	Syphilis	—	—
17	1936	Little	—	—	—	Chronic osteomyelitis	—	—
18	1936	Sinberg	M	15	Manubrium	Echinococcus	Drained	Recovered
19	1946	Kinsella	M	74	Gladiolus	Tuberculosis	—	—
20	1946	Kinsella	F	42	Manubrium	Tuberculosis	—	—
21	1946	Dundon, Williams, and Laipply	F	17	Gladiolus	Eosinophilic granuloma	X-ray three times	Improved: three recurrences: still has mediastinal mass

Before undertaking this extensive resection of the sternum, considerable thought was given to the problem of stabilization of the chest wall and the necessity for this in a young, healthy, robust individual of very active physical habits. Certainly a chest wall defect of the size produced by excision of this tumor would leave a large defect which, if unrepaired, though covered by skin and subcutaneous tissue, would permit rather violent paradoxical motion of the underlying pleura and mediastinal structures, particularly under effort, and would afford no bony protection for the underlying heart and great vessels. Both of these would be a distinct handicap and hazard to a young man leading an active life. Partial stabilization of the defect by a massive fascial transplant was considered but rejected as inadequate to provide the desired stability. The possibility of providing a pad and metal plate to protect this area was likewise rejected as unsatisfactorily.

The desirability of providing bony support to this portion of the chest was appreciated. The possibility of using rib for reconstruction of the sternum was considered, but realizing the difficulties of fitting the curved pieces in such a manner as to provide immediate as well as late stability, it was considered undesirable except as a source for bone chips later used in the reconstruction. A tibial bone graft was decided upon as the best source of bone which could be fitted accurately and fixed to give the desired support immediately, as well as to provide a scaffold to which new bone could become attached, in spite of the extra operating time which would be required in procuring and shaping it to the desired proportions. The use of a second operating team to carry out this work simultaneously with the work would have aided very materially, but during wartime this was not available, so all the work was carried out by one surgical team. The type and character of the reconstruction are best illustrated by the schematic drawing, Fig. 5, Plate I.

OPERATIVE PROCEDURE

The operation was carried out on May 4, 1945, at St. Mary's Hospital with the patient under pentothal-ethylene intratracheal anesthesia administered by Dr. William A. Friend. A wide segment of the left posterior ninth rib was resected subperiosteally and the bone saved for future use as chip grafts. The wound was closed and the patient turned onto his back. The right shin was prepared, and from the anterior tibial surface a large bone graft 2 cm. wide and 25 cm. long was removed with an electric saw, and preserved in a sterile manner for future use in reconstruction of the chest wall. A long, transverse incision was made at the level of the third interspace anteriorly extending from the left to the right nipple line. The skin flaps were dissected up to expose the sternal mass from top to bottom.

A vertical incision was then made in the midline through the fascia of the pectorals, and this fascia and the pectoral muscles were dissected off the tumor mass and retracted laterally. A piece of the fourth costal cartilage on the right and the left sides was then resected subperichondrally, and the posterior perichondral bed was incised to admit the finger for dissection of the pleura from the mass and the chest wall beyond the margins of the sternum. The costal cartilages of the second to the seventh ribs on both sides were then cut parallel to the border of the sternum about 2 cm. lateral to the tumor. In doing so, the internal mammary vessels were injured on both sides, necessitating ligation. By finger dissection, the posterior aspect of the manubrium was freed and found to be without tumor involvement above the manubriosternal junction. A Gigli saw was passed underneath the manubrium, which was then cut across about 2 cm. above the tumor.

The tumor mass was then freed carefully by finger and gauze dissection from the underlying pleura, and retracted toward the left. In so doing the right pleural cavity was opened and a small pneumothorax developed. This leak was controlled by digital pressure until the right pleura was completely free from the tumor, when the lung was completely expanded by the anesthetist and the pleural defect closed with a fine chromic catgut suture on an atraumatic needle. Dissection of the pleura from the posterior aspect of the tumor was somewhat difficult, as the pleura was quite adherent, although there was no evidence of invasion of the tumor beyond its shell. With the right pleural cavity closed, the dissection was then carried toward the left where the tumor was freed from the left pleura, but in so doing the left pleural cavity was also entered in one small area. Following the removal of the tumor the left lung was expanded by the anesthetist and the pleural defect was closed in a manner similar to that used on the right. After removal of the tumor mass we were confronted with a large anterior chest wall defect through which the respiratory and pericardial activities were quite marked.

The stability of the chest wall was then re-established by using the tibial bone graft previously removed in the following manner. The excised bone graft was split longitudinally with a motor saw, leaving two strips, each 1 cm. wide, which were then cut to the desired length to reach from the lower end of the cut manubrium to the severed seventh cartilage and xiphoid below. These were fitted to either side of the defect in vertical position and marked across at the level of each cartilage. Two holes were then drilled through each vertical bone graft in a position to correspond to each cartilage as well as to the manubrium at the upper end. The bone plates were then put into position and fixed to the manubrium above and to each abutting cartilage with mattress sutures of chrome wire. Two cross pieces of the bone were then fitted transversely between the vertical two as the rungs of a ladder, and wired into position at the levels of the third and fifth anterior cartilages. This afforded a rigid ladderwork and gave a perfectly stable chest.

The mediastinal structures posterior to the ladder were then approximated to it with interrupted chromic catgut sutures in order to obliterate the dead space. All remaining pieces of tibial bone as well as the excised portion of the ninth rib were then cut up into fine bits which were sprinkled loosely into the defect between the ladder, and 2½ Gm. of sulfanilamide were sprinkled over this area. The pectoral muscles and fasica were then approximated at the midline by a series of interrupted and continuous chromic catgut sutures and the fascial layer pulled down to the bone ladder by transfixing it with the long ends of the catgut sutures previously used to bring up the mediastinal structures against the posterior aspect of the ladder. A needle was then inserted through the muscle flaps, all air was aspirated and 100,000 units of penicillin in saline solution were injected. The skin flaps were then brought down, the subcutaneous fascia closed with interrupted plain catgut sutures and the skin with a running dermal suture. A small pad was placed over the incision and a small aluminum splint strapped down over the sternal region with broad straps of adhesive in order to obliterate all dead space.

The patient tolerated the procedure very well and was given 3,000 c.c. of blood, 250 c.c. of plasma, and 400 c.c. of normal saline while on the table, and was returned to his room with a blood pressure of 110/85 mm. mercury. His postoperative condition was quite satisfactory. During the first three days there was some febrile reaction due to sputum retention which was relieved by spontaneous cough. There was some oozing from the site of the tibial graft. Healing occurred by primary intention in all wounds except for one small area of skin necrosis apparently from interruption of the local skin blood supply in dissecting the flaps. The patient was continued on penicillin for thirteen days with a total of 980,000 units being given. Sulfadiazine was also administered for a period of nine days.

He was discharged from the hospital on the twenty-seventh postoperative day, ambulatory, on crutches, with a short leg brace fitted to the right lower extremity to protect this member from fracture because of the weakening produced by the very large bone graft which was removed. The chest wall was stable from the start and has remained so with no evidence of paradoxical chest wall motion. The patient was able to cough and expectorate spontaneously from the day of operation although there was considerable associated pain. While subsequent x-rays showed a breaking of a number of the wires used in the reconstruction of the sternum, the bone transplants remained in place and apparently served their purpose. The short leg brace was worn for twelve weeks as a precautionary measure. The patient resumed his activities almost at once, returning to full work within three months of the time of his surgery, then taking up flying and acquiring a private flying license within six months of his operation. He has remained in good health carrying on activities of all types.

Pathological report.—Grossly, the specimen consists of a tumor mass in the body of the sternum. The tumor measures 2½ inches in width, 2¼ inches in thickness, and is 5 inches in length. The tumor is a uniform oval mass with almost no lobulation. A thir

Plate I.—A. C. K. Aged 33. Benign giant cell tumor of the body of the sternum.

Fig. 1.

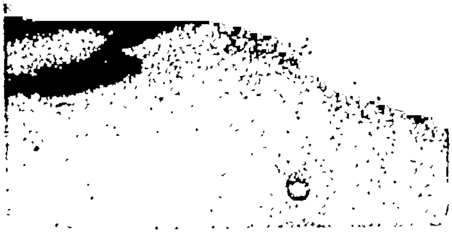


Fig. 1.—Preoperative tumor mass.

Fig. 2.—Lateral x-ray film of tumor.

Fig. 3.—Oblique x-ray film of tumor.

Fig. 2.

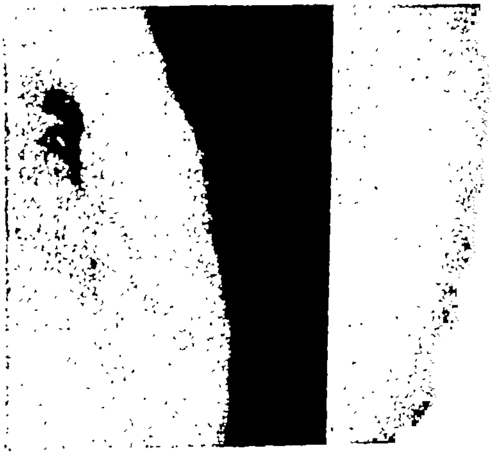
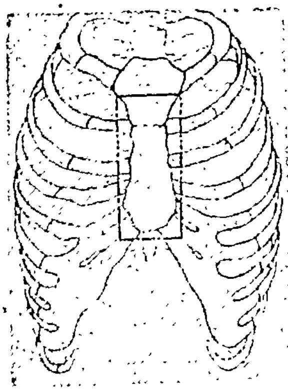


Fig. 3.

Fig. 4.



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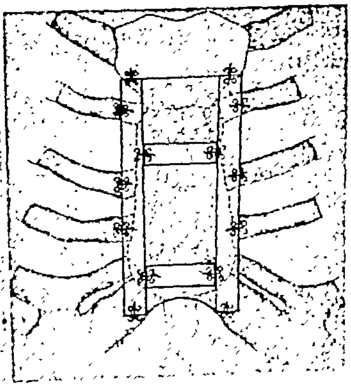


Fig. 6.

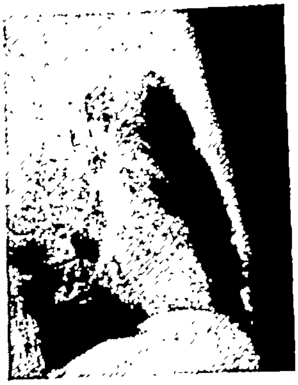


Fig. 4.—Chart showing extent of resection of the sternum.

Fig. 5.—Drawing of reconstruction of the sternum by tibial bone graft and bone chips.

Fig. 6.—Late postoperative lateral x-ray of reconstructed sternum.

Plate II.—A. C. K.

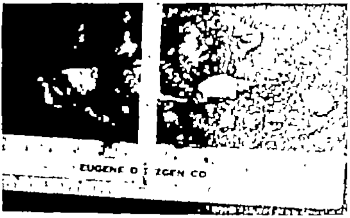


Fig. 1.

Fig. 1.—Photograph, lateral view of resected tumor.

Fig. 2.—Sternal tumor split longitudinally.

Fig. 3.—Postoperative photograph of chest one year following surgery.

Fig. 4.—Photomicrograph of benign giant cell tumor of the sternum, low power.

Fig. 5.—Photomicrograph of benign giant cell tumor of the sternum, high power.

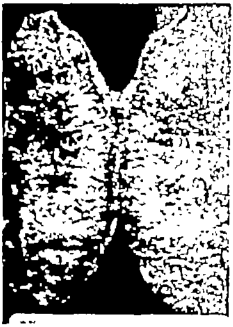


Fig. 2.

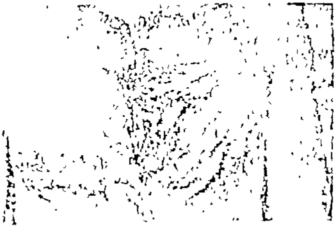


Fig. 3.

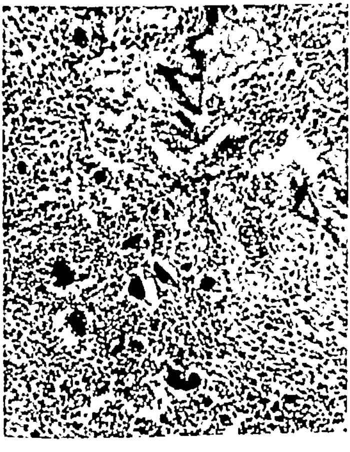


Fig. 4.

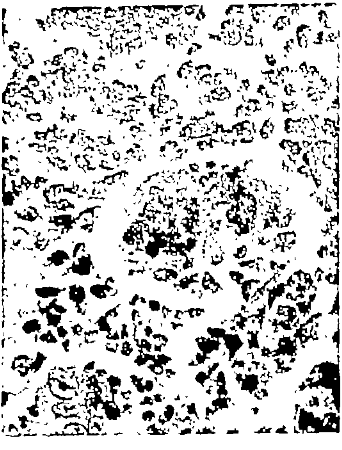


Fig. 5.

shell of bone, averaging about 3 mm. in thickness, forms a capsule for the tumor. No perforation through this shell of bone can be found. The tumor is solid, firm, and pink in color, and has a fibrous texture.

Microscopic examination shows that the tissue is almost entirely fibroblastic in structure. Imbedded in the fibrous matrix are large numbers of giant cells. There is no suggestion of malignancy. Dr. E. T. Bell and Dr. A. C. Broders independently interpreted the tumor as a benign giant cell tumor.

This diagnosis was subsequently certified by the Tumor Registry of the American Institute of Pathology, AIP Accession #168780.

Late postoperative x-rays of the chest on July 13, with detail films of the region of the sternum and films of the right leg showed a linear defect in the middle and proximal third of the tibia, extending slightly into the distal third and representing removal of a large segment of the anterior portion of the tibia for transplant into the region of the sternum. There was evidence of new bone formation at the site of the removal of the bone from the tibia, where a partial reconstruction had taken place and there was no evidence of any complication at the site of the tibial resection. Two linear fragments of bone were demonstrated in the somewhat V-shaped position in the region of the sternum, with two smaller crossbars shown in the upper and lower portions of the sternal region. Multiple wire loops were shown in this region with some of these apparently connecting the larger transplant to the costal cartilages while others connected the crossbars to the main transplant. Some of these wires had been broken. There was some overlapping of the crossbars over the main transplant; the wire loops had become disengaged. A lateral film, however, showed good alignment of the transplant in this direction. It was impossible to determine the amount of new bone formation. Partial resection of the left ninth rib was demonstrated. The lungs were otherwise clear.

An extensive search of the literature and a review of collected series reveal authenticated reports of giant cell tumors involving many bones of the body, but thus far we have been unable to find any mention of giant cell tumor involving the sternum. It may be that this is the first authenticated case of giant cell tumor of the gladiolus sterni. The question may now be raised that had we known that this was a benign giant cell tumor of the sternum, should we have treated it by x-ray, by curettage, or by resection? These questions were debated before the surgery was undertaken. It was then and it is now our opinion, that in this instance surgical excision and reconstruction of the sternum would be a far more satisfactory answer to the problem than either of the other methods. With the uncertainties of diagnosis which were present at the time, it presented a very satisfactory answer to the problem.

CASE 2.—G. N. D., aged 60, a white man, president of a department store, was first seen in consultation Feb. 15, 1946, with Dr. S. Marx White because of a tumor mass involving the manubrium sterni. His past history was negative except for a slight chronic arthritis, during the study of which some dental apical abscesses and a mild, chronic, nonspecific prostatitis were treated as possible foci. For a number of years he had also had moderate, rather labile hypertension without symptoms.

Present complaint.—For the previous two years there had been a gradual development of a rounded, projecting tumor mass from the manubrium sterni, which the patient thought was due to coughing. There was no history of trauma and no pain or tenderness. The growth was slow and gradual for the first ten months with slight if any growth since that time. There had been no weight loss. The tumor was beginning to interfere with bending his head forward and with the fit of his shirt collar.

Physical examination revealed a well-nourished, well-developed adult white man presenting no abnormality except for hypertension 206/106 mm. of mercury, with a corresponding slight cardiac enlargement. His hypertension was not constant, as at times the pressure was found as low as 170/90 mm. of mercury. There was a smooth mass about 8 cm. in diameter projecting anteriorly from the manubrium sterni. It was bony hard in character and fixed in position. There was no pulsation, no tenderness, and no skin change. The skin and subcutaneous tissues moved freely over it. The clavicles and sternoclavicular joints apparently were not involved, nor was their motion restricted.

The hemoglobin was 12 Gm., white blood cells 8,250, with 67 per cent polymorphonuclears III and 2 per cent polymorphonuclears II, 26 per cent lymphocytes, 4 per cent mononuclears, and 4 per cent eosinophiles. The bleeding time was one minute and fifty seconds. The blood calcium was 7.00 mg. per 100 c.c. on Jan. 15, 1946, and 8.9 mg. on January 30. The blood phosphorus was 2.6 mg., blood phosphatase 2.6 mg., and the total protein 8.6 Gm. per 100 c.c. of blood. Urinalysis revealed a pH of 7.5, specific gravity of 1.014, albumin two plus, sugar negative, two to three leucocytes and one to three red blood cells per high power field on one examination, but none subsequently. There were no casts present. Examination for Bence-Jones protein showed a questionable faint trace on one examination, but four subsequent tests were negative.

X-ray films of the chest showed increased bronchovascular markings throughout of a diffuse type suggestive of bronchitis, with some calcification of the aorta. There was a slight cardiac enlargement of a hypertensive type. The ribs presented no evidence of disease. X-ray examination of the sinuses revealed that the right antrum was cloudy and the right frontal sinus somewhat hazy. The skull showed no evidence of bone destruction but there was a slight granular appearance with uneven calcification suggesting some disturbance of the calcium-phosporus metabolism. X-ray studies of the pelvis showed some phloboliths on both sides with slight possible increased density in the bones on the right side. An x-ray film of the knee was negative. Intravenous urograms showed both kidneys, pelvis, and ureters well visualized, and they appeared to be entirely normal. The calices of both kidneys were somewhat elongated, but this appeared to be on a congenital basis and did not suggest renal tumor. The spine showed a fairly marked hypertrophic arthritic change and a slight scoliosis of the lumbar spine toward the left.

Roentgenograms of the sternum in multiple views taken in the oblique and lateral projections showed a multiloculated, expanding tumor involving the manubrium, without involvment of the body of the sternum. The normal pattern of the manubrium was replaced by a cystic type of pattern with multiple irregular lines of density which apparently represent the outlines of small cysts. The manubrium was expanded both in the mediolateral and anterior-posterior direction, but apparently the outer margins of the manubrium were not destroyed. The findings indicated a multiloculated, cystic type of tumor expanding the manubrium but without definite destruction of any of the vertical margins.

The diagnostic problem here was not simple. Because of the slow growth and apparently static course in recent months, a benign tumor was suspected. Careful clinical search revealed no primary tumor elsewhere which might give rise to metastases to the sternum. There were no nodules in the thyroid gland and urograms revealed no evidence of distortion or displacement suggestive of renal or adrenal primary tumor. In addition, the tumor did not pulsate. The possibility of myeloma was considered, but it was felt that the slow progress of the tumor was against this, and the negative findings in the ribs, skull, and other bones was definitely against such a diagnosis. The absence of Bence-Jones protein in repeated examinations of urine was also against such a diagnosis. There also was the fact that the x-ray studies revealed evidence of bone deposit at the periphery of the tumor rather than complete destruction of the areas involved. The possibility of hyperparathyroidism was also considered because of the somewhat cystic nature of the tumor suggested by the x-ray studies, but the normal blood calcium and phosphorus values were considered to rule out such a condition. We felt, therefore, that we were dealing with a primary tumor of the sternum and that its local characteristics and duration definitely favored a benign type of tumor. With the tentative diagnosis of benign neoplasm of the manubrium sterni, surgical excision was recommended.

Preoperatively, considerable thought was given to the necessity or desirability of providing some reconstruction for the portion of sternum and inner clavicles which we planned to remove. This could have been accomplished rather easily by the use of bone graft fitted to rebuild the manubrium with a transverse piece above interposed between the two resected clavicles. It was decided, however, that the amount of handicap which will follow resection of the manubrium without reconstruction would not be great because of its relatively small size, and that fixation of the clavicles to each other without an intervening joint could well prove a far greater handicap to the individual than leaving the inner ends entirely free. Recognizing that arm and shoulder motion can be quite adequate without the clavicles, it was decided to make no attempt at fixation of the inner clavicle, relying upon the lateral insertion of the sternocleidomastoid above, the pectoral muscles below, and the subclavius underneath to stabilize the inner end. The correctness of this judgment has been borne out by the lack of handicap or embarrassment and a satisfactory arm and shoulder motion presented by the patient during the year following his operation.

OPERATION

On March 7, 1946, under sodium pentothal, nitrous oxide oxygen anesthesia with curare (administered by Dr. R. T. Knight) with the patient in dorsal recumbent position, a long transverse incision was made at the level of the third rib extending from nipple line to nipple line. The skin and subcutaneous tissues were reflected upward and downward as flaps. A vertical incision was then made in the midline through the pectoralis fascia and muscle, which were separated by cutting them a short distance from their origin from the upper sternum and retracting them laterally. A segment of the inner end of the right and left third cartilages were then resected subperichondrally. The perichondrium was then cut across and by finger dissection the posterior aspect of the sternum was dissected free across the midline at the level of the second interspace. A Gigli saw was passed through this area and the sternum was cut transversely at the lower end of the second interspace. Dissection was then carried upward underneath the sternum separating the mediastinal tissues from their attachment to the tumor. The posterior aspect of the tumor was somewhat nodular and hard but there seemed to be no infiltration into adjacent tissues. The internal mammary vessels were ligated on both sides, and the second and first cartilages were freed and cut across with a laminectomy shears because of calcification and a bony shell which extended out, particularly over the first.

The sternal and a portion of the clavicular attachment of the sternocleidomastoid was then separated, leaving some muscle attached to both bones. The inner 3 cm. of the right clavicle was then cut off with a Gigli saw, leaving it attached to the tumor mass. The tumor was then rotated to the left, the sternothyroid and sternohyoid muscles on both sides were clamped and cut close to their attachment to the sternum, and the left clavicle, the first and second cartilages were treated in a manner similar to the right. Very small leaks were created in both pleurae but the lungs were kept well expanded at all times. All visible defects in the pleura were sutured with fine catgut on atraumatic needles but minute leaks still persisted. As there appeared to be no excessive motion of the mediastinal structures and because of the small size of the defect, it was decided not to reconstruct the sternum.

After control of all bleeding, the pectoral muscles were approximated across the midline with interrupted, followed by continuous, chromic catgut sutures, and the cervical fascia, and the lower ends of the sternocleidomastoid and prethyroid muscles were sutured down to the upper edge of this approximated muscle. A small catheter was inserted through the muscle and into the mediastinal space, all air and fluid aspirated, and 100,000 units of penicillin instilled into the wound. The skin flap was then brought down and tacked to the underlying muscle, the subcutaneous fascia closed with interrupted plain catgut sutures, and a lock stitch of dermal suture placed in the skin. A small drainage catheter was left in place under the skin flap for aspiration of fluid as it formed and a pressure dressing was applied. The patient tolerated the extensive procedure very well. He received 2,000 c.c. of blood on the table and was returned to his room in good condition.

His postoperative condition and hospital course was quite uneventful except for some sputum retention and local atelectasis which developed on the third day and was relieved by the use of steam inhalations and hammer percussion over the involved lobe. Sulfadiazine and penicillin were continued postoperatively for seven days. The wound healed without difficulty except for a small accumulation of serous fluid under the lower flap which was aspirated on three occasions postoperatively. A pressure dressing was maintained under adhesive strapping for the first week and was then discarded. Free arm motion was permitted from the start and the patient was encouraged to move about and do things by himself. A high back rest was used after the first twenty-four hours and active leg exercises were done from the beginning. The patient was permitted to sit up on the third day, and was out of bed on the fifth day in a chair at the bedside. We then encouraged him to move about and walk, which he did daily until his discharge from the hospital on his tenth postoperative day. He resumed his active work at the office in approximately a month from the time of his surgery and within two months was driving his car and carrying on all of his usual activities. Immediately postoperatively, and for the first few weeks after the surgery, there was considerable induration in the region previously occupied by the manubrium and inner clavicle, giving a firm chest wall and little or no motion. Gradually, however, as the infiltration from operative manipulation, and suturing subsided, the tissues became softer and a moderate amount of pulsation and paradoxical motion could be observed, but at no time was this distressing or disturbing. There was no tendency for the head to be pulled forward by the shortened sternocleidomastoid or prethyroid muscles, nor to drop backward because of any laxity of these muscles. No support was provided for the shoulders at any time, nor was any necessary, as no disability or instability resulted from resection of the inner clavicles

Pathologic Report.—Grossly the specimen consists of the manubrium of the sternum, *about 2 cm. of the adjacent ends of the clavicles, and a thin segment of the body of the* sternum. The manubrium is expanded by a roughly oval tumor which measures 5½ by 8 cm. and is 4 cm. in thickness. There is a thin shell of bone over the tumor and in small areas the bone is entirely destroyed and the tumor is restrained only by periosteum. There is no direct infiltration into the adjacent tissues and no tumor is visible in the short segments of the clavicles. The surface of the mass is slightly lobulated. The interior is soft, solid, and can be disintegrated readily between the fingers. The tumor is irregularly mottled by reddish and white areas. An occasional spicule of bone remains in the interior.

Microscopic study shows extensive necrosis and disintegration by hemorrhage. The viable tumor is very vascular. There is a loose, spongy, fibrous stroma and the spaces are filled with cells having striking uniformity. The cells are cuboidal and have prominent nuclei without mitotic figures. Some nuclei are eccentric.

This diagnosis of myeloma was subsequently verified by Dr. E. T. Bell, Dr. A. C. Broders, and Dr. E. Schleicher. Later this diagnosis was certified by *the Tumor Registry of the* American Institute of Pathology, Accession No. 108799, as a solitary plasma cell myeloma of the sternum. Clinically there would appear to be sufficient evidence to classify it at least *temporarily as a solitary myeloma.*

DISCUSSION

In retrospect, several interesting questions present themselves: (1) Could this tumor have been diagnosed as myeloma preoperatively? (2) What additional evidence could have been obtained to aid in diagnosis? (3) Should it have been excised? (4) Should a reconstruction of the sternum been carried out? (5) Is this tumor merely a precursor of multiple myeloma?

The possibility of myeloma was considered before surgery was undertaken, but was ruled out by the long course, the presence of some bone proliferation at the periphery of the tumor, the absence of lesions at the favored myeloma sites, that is, skull, pelvis, and ribs, and the absence of Bence-Jones protein in the urine. He presented none of the six cardinal diagnostic points listed by Geschickter and Copeland as suggestive of multiple myeloma, except his age.

Plate III.—G. N. D. Aged 60. Solitary plasma cell myeloma of manubrium.

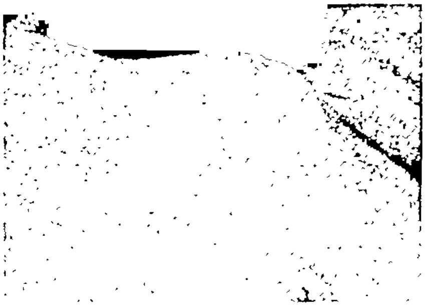


Fig. 1.

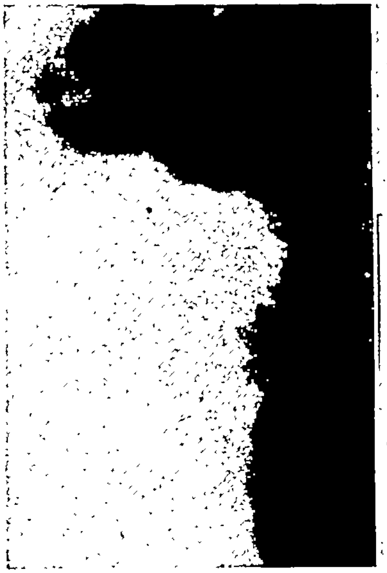
Fig. 1.—Preoperative photograph of tumor.

Fig. 2.—Oblique x-ray film of tumor showing trabeculation and intact peripheral bony shell.

Fig. 3.—Lateral x-ray film of tumor.

Fig. 4.—Resected manubrium with attached inner ends of clavicles.

Fig. 5.—Manubrium split longitudinally showing interior of tumor and intact sternum below.



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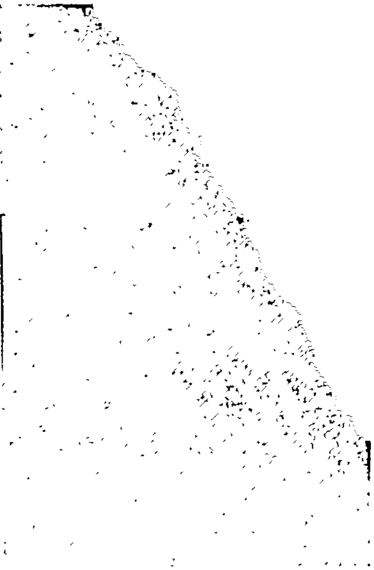

Fig. 3.

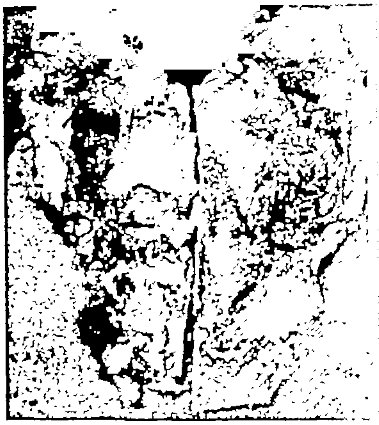

Fig. 4.

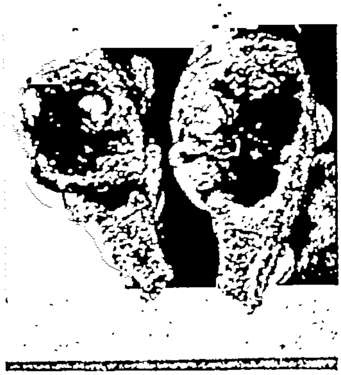

Fig. 5.

Plate IV.—G. N. D.

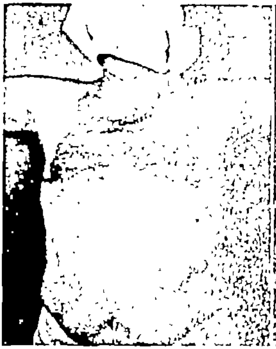

Fig. 1.

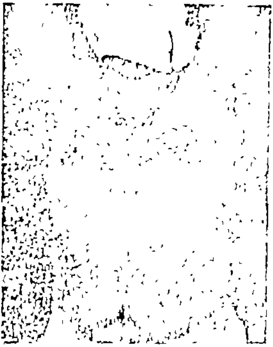

Fig. 2.

Fig. 1.—Postoperative photograph of chest [illegible] operation showing depression at site of resected manubrium and prominence of [illegible] clavicles.

Fig. 2.—Same, showing free arm motion.

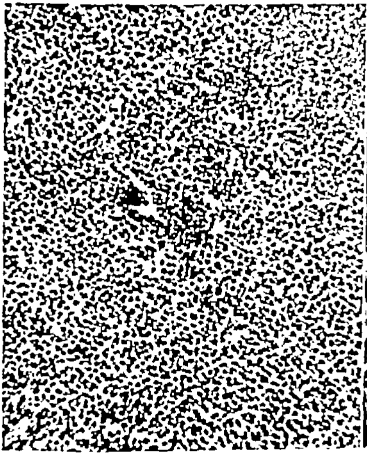

Fig. 3.

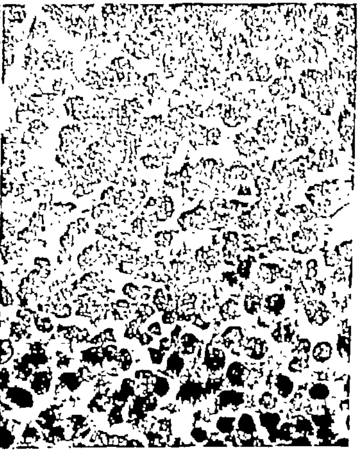

Fig. 4.

Fig. 3.—Photomicrograph of plasma cell myeloma, low power.

Fig. 4.—Photomicrograph of plasma cell myeloma, high power.

Biopsy of the tumor certainly would have established the diagnosis, but was not done because we did not wish to risk dissemination of an apparently localized process. A sternal bone marrow biopsy might possibly have given a clue, but this is very doubtful at the stage seen. The bone marrow of the clavicles revealed no evidence of myeloma. The blood bank records show that considerable difficulty was encountered in preparing blood for transfusion for him because of pseudoagglutination. His serum agglutinated cells from all donors, and also his own cells both in the cold and at 37°C. No abnormal cells were observed in the blood smears. The total blood protein was 8.6 Gm. per cent, but no fractionation was done. The sedimentation rate was not recorded.

The advisability of resection under the circumstances cannot seriously be questioned. For benign and primary malignant tumors, primary resection was certainly the procedure of choice. The tumor itself was producing trouble enough to justify its removal. Even for a known, apparently solitary myeloma of a site suitable for complete excision, operative removal would seem to be the logical method of attack.

Reconstruction of the excised manubrium and inner clavicles was not carried out on this patient. The loss of the inner clavicles has been no serious handicap, as orthopedic surgeons have known for some time. Stabilized above by the clavicular insertion of the sternocleidomastoid, below by the pectorals, and posteriorly by the subclavius muscles, the free ends do not project unduly, or have excessive motion on exertion. There has been no tendency for the shoulders to drop forward, and shoulder braces, as advocated by some, have been unnecessary.

Soon after the operation, induration in the area of the resected manubrium gave a firm support to the soft tissues covering the underlying pleura and vessels, so that no unusual motion occurred. Subsequently, with absorption of the inflammatory reaction, increasing motion appeared with inspiratory retraction and bulging on straining, which, though not a handicap to a man of his age and occupation, could have been a hindrance to a young man performing active physical labor. Reconstruction could have been accomplished with a fascia lata transplant, or better by a bone transplant with suture of the cervical fascia to the bony framework.

The question may well be asked if this tumor is truly a solitary myeloma or merely a local tumor, a precursor of multiple myeloma. Only time will answer this question. To date, one year after resection and three years after onset of the tumor, the patient enjoys good health with no complaints of any sort suggestive of generalization. His urine still remains negative for Bence-Jones protein, and blood specimens reveal no abnormal elements. X-ray studies have not been made. Two abnormal laboratory findings, neither clearly diagnostic but either suggestive of some disturbance, have been recorded. The sedimentation rate is extremely fast, 122 mm. in one hour, with the serum albumin-globulin ratio disturbed, serum albumin 4.116 and serum globulin 4.204 Gm., though the total protein level falls within normal limits. Their true significance is unknown.

Opinions differ widely on the problem of solitary myeloma. An editorial in the *Journal of the American Medical Association* in 1935 concluded with the statement, "It appears, therefore, that besides the generalized form of myeloma with its hopeless outlook, there is a localized form amenable to surgical therapy and offering a favorable prognosis." Cutler, Buschke, and Cantril, in 1936, reported on eighteen collected cases of which five growths solitary at first, later became generalized. The other thirteen growths revealed no generalization during the period of observation. Seven patients, however, had been followed for less than nineteen months. Two were alive and well, four and ten years after onset. Pasternack and Waugh, in 1939, reported one patient alive and well for 7½ years. King, in 1940, records one patient with solitary intraosseous myeloma which became generalized four years later. Tennent in 1945 collected 49 cases of solitary plasmocytoma with one of his own, and records 30 per cent developing multiple myeloma in from two months to six years, although only 50 per cent had been followed more than two years. Bayard and Heck in 1947 reporting on the Mayo Clinic series of eighty-three proved cases of multiple myeloma, report ten patients with local tumor formation, but also report that since doing sternal biopsies, no instances of solitary myeloma have been recorded.

It would seem that the apparently localized and solitary type of myeloma will be found less frequently if complete studies are made, that the prognosis is somewhat less unfavorable than the multiple type, that surgical excision of resectable lesions is justifiable, but that the eventual result can be determined only by time. The effect of some of the newer drugs now being tried in the treatment of myeloma will be watched with interest in the hope that more permanent results may be obtained.

SUMMARY

Two unusual sternal tumors, a benign giant cell and an apparently solitary plasma cell myeloma, are presented, and surgical treatment outlined. The available literature on benign, malignant, and metastatic tumors and some of the inflammatory lesions of the sternum are reviewed and tabulated.

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PULMONARY EDEMA

EXPERIMENTAL OBSERVATIONS ON DOGS FOLLOWING ACUTE PERIPHERAL BLOOD LOSS

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INTRODUCTION

THE purpose of this paper is to present a series of observations on dogs which give evidence that acute peripheral blood loss will produce pulmonary pathologic change and circulatory imbalance; that these pulmonary changes are those of edema, hemorrhage (Fig. 1) vascular congestion, and endothelial damage, with the production of a block to normal circulation; and that, because of the stasis in pulmonary circulation which results from these changes, there is developed an acute systemic circulatory imbalance in which arterial pressures are low and venous pressures are high (Fig. 2). The lung changes which follow peripheral blood loss are not transient; they have been observed to last as long as five days, and to be aggravated by intravenous therapy if not wisely administered as to type and quantity.

It seems probable that the physiologic changes involved in acute hemorrhage and so-called shock are similar if not identical, and that each of these conditions is characterized by acute decline in blood pressure, loss of blood volume, slowing of circulation, anoxia, capillary congestion, increase in capillary endothelial permeability, tissue edema, and cardiac insult (Fig. 8). In acute hemorrhage, this physiologic chain reaction is touched off by a loss of blood volume, whereas in shock the same series of events may be triggered by a cardiac insult or by a mechanism leading to reduction of effective blood volume.

It must be pointed out that these experiments are affected by individual ability of the experimental animal to withstand and compensate for blood loss, and that, as in humans, age and physical fitness seem to be factors in the variable findings which result. It seems justifiable to present the results of these experiments because fundamental principles are involved which may have practical application to surgery, postoperative care, and the prevention of postoperative pulmonary edema in man.

PART ONE

OBSERVATIONS ON PULMONARY ARTERIAL PRESSURE AND PERIPHERAL VENOUS PRESSURE FOLLOWING ARTERIAL BLOOD LOSS

During experimental studies of the lymph flow from the right thoracic duct of dogs, some interesting pulmonary findings were made.[1] In response to the acute loss of 25 per cent of blood volume from the femoral artery, a stimulation

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of pulmonary transudation was evidenced by an immediate increase in lymph flow from the pulmonary lymphatic duct. Histologic examination of the lungs of these animals showed evidence of pulmonary edema, capillary congestion, and hemorrhage. The concept that peripheral arterial blood loss could produce pathologic change within the lung aroused our curiosity and led to further investigation in pulmonary physiology and pulmonary fluid economy during and following hemorrhage.

A study of pressure changes in the peripheral vascular circulation seemed desirable when it was apparent that so important an organ in circulatory mechanism as the lung was being damaged by the acute loss of peripheral arterial blood. Continuous peripheral venous and peripheral arterial pressures were then recorded on the kymograph during and following hemorrhage, and it was noted that following an initial pressure fall of both these systems there was a period of instability of the venous system in which there was a marked elevation in pressure from the femoral vein without corresponding pressure elevation in the femoral artery. This observation of an impounded pressure in the peripheral venous system seemed to be evidence of circulatory obstruction developed either within the pulmonary circuit or within the right or left heart. A study was undertaken to ascertain pressures within the pulmonary artery under these same circumstances of hemorrhage. This evidence seemed a requirement, for if the right heart could be eliminated as a cause of this impounded venous pressure following blood loss, then pressure changes within the pulmonary artery and pulmonary arterial capillary bed could be considered to be evidence of a mechanical factor involved, in which damage to the endothelium of the pulmonary capillary bed may be augmented by insult to the functional ability of the left heart.

The results of this experimental work have been reported more completely in an earlier paper.[1] The findings described were consistently observed in seven consecutive experiments in which pulmonary arterial pressures and peripheral venous pressure were recorded, four with the Wiggers optical manometers (Fig. 3) and three with the kymograph (Fig. 4).

Following withdrawal of 20 per cent of blood volume (5 per cent loss allowed for operative procedure) an immediate drop of pulmonary arterial pressure, systemic arterial pressure (femoral artery), and peripheral venous pressure (femoral vein) occurred, as might be expected. In the interval of ten to fifteen minutes following blood loss, a slight but perceptible rise in all pressure was noted. From twenty to twenty-five minutes later the pulmonary arterial pressure increased progressively; from forty to forty-five minutes after blood loss, this pressure often reached or surpassed its previously recorded normal level; and after about forty minutes of such elevated pressure the pulmonary artery pressure fell back to levels comparable to the low systemic arterial pressure which was being maintained.* Examination of Fig. 4, which is a copy of a section of a kymogram, shows the marked increase in pulmonary artery pressure which is maintained for an hour or more after peripheral arterial blood loss, and Fig. 3 shows this same change recorded photographically with a Wiggers manometer.

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*On several occasions larger quantities of blood were removed and fatal shock level depression of arterial pressure produced. In each case the pulmonary artery pressure and the peripheral venous pressure continued at excessively high levels, in fact, until the vasomotor-collapse of death, these pressures were elevating precipitously while peripheral arterial pressures steadily declined.

At the same time, peripheral venous pressure began rising ten to twenty minutes after pulmonary arterial tension started its rise, and usually reached its previously recorded normal level an hour or more after blood loss. Peripheral venous pressure increase has always shown this lag behind the rise in pulmonary arterial pressure. This interval may be the time required for pulmonary artery impounded pressure to produce right ventricular dilatation and incompetency, and a spilling over of pressure into the peripheral venous system.

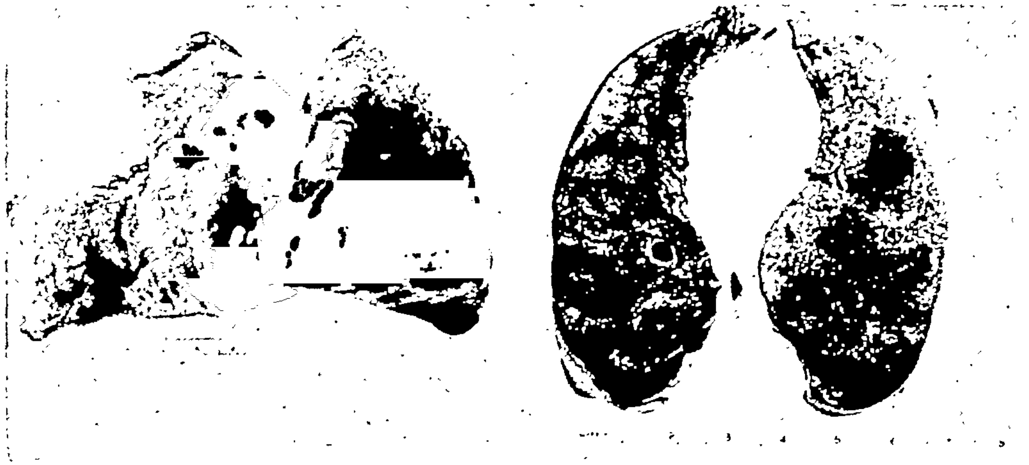


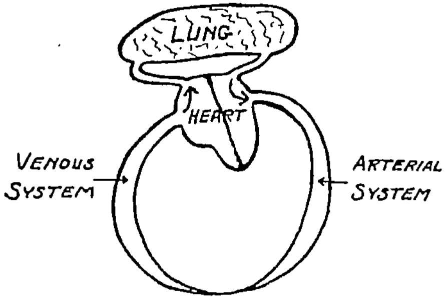
Fig. 1.—Photograph of lung following hemorrhage with two views showing pleural and cut surfaces. *A*, right lung of a dog in which a loss of 25 per cent of blood volume was sustained from the femoral artery four hours before sacrifice. Note dark mottled areas of hemorrhage throughout the lower lobe and the lower portions of the upper and middle lobes. *B*, sections through right and left lower lobes showing extensive hemorrhage throughout the right lower, and in portions of the left lower lobes.

The normal relationship between the arterial and venous systems was temporarily upset by the changes produced by acute blood loss, and the pressure change in the venous system was much greater proportionally than that within the systemic arterial system. The evidence of an increase in pulmonary artery pressure following acute blood loss seemed to be good proof that a circulatory obstruction was produced within the lung itself and aggravated by some failure of the left heart. Indeed, changes in pulmonary artery pressure reflect changes throughout the entire pulmonary capillary bed. A rise in pulmonary artery pressure is indicative of a rise within the pulmonary arterial capillaries and vice versa. This system is a relatively short closed one with the pulmonary valve at one end and the capillary tufts at the other, and pressure alterations within the lung capillary bed would quickly be indicated in the pulmonary artery.

CONCLUSION

1. Peripheral arterial blood loss results in circulatory imbalance characterized by low peripheral arterial pressure and a transient elevation of both pulmonary artery pressure and peripheral venous pressure.

2. An impounded pressure within the pulmonary artery following peripheral blood loss is evidence that an obstruction to normal circulation exists within the lung, the left heart, or both.



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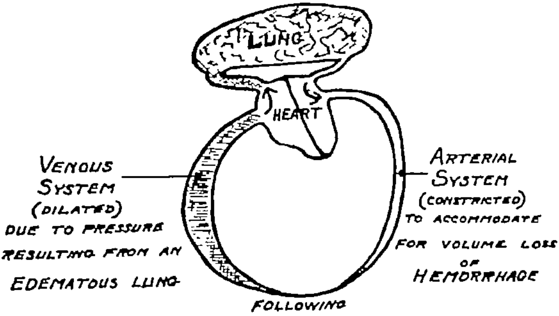
LUNG
HEART
VENOUS SYSTEM (DILATED) DUE TO PRESSURE RESULTING FROM AN EDEMATOUS LUNG
ARTERIAL SYSTEM (CONSTRICTED) TO ACCOMMODATE FOR VOLUME LOSS OF HEMORRHAGE

FOLLOWING HEMORRHAGE

Fig. 2.—Schematic drawings to show the changes which take place within the vascular system when there is acute peripheral arterial blood loss.

Part Two

Quantitative Changes in Pulmonary Moisture Following Acute Hemorrhage

Data have been submitted (Part One) which show that circulatory imbalance is produced following acute peripheral arterial blood loss and that this condition is characterized by a temporary elevation of both pulmonary artery and peripheral venous pressures without corresponding rise of pressure in the peripheral arterial system. It was suggested that the obstructive mechanism producing this circulatory imbalance following acute peripheral blood loss must be within the pulmonary capillary bed, the left heart, or both. This conclusion was based upon the fact that a period of elevated pressure was observed to exist within the pulmonary arterial portion of the venous system, while no

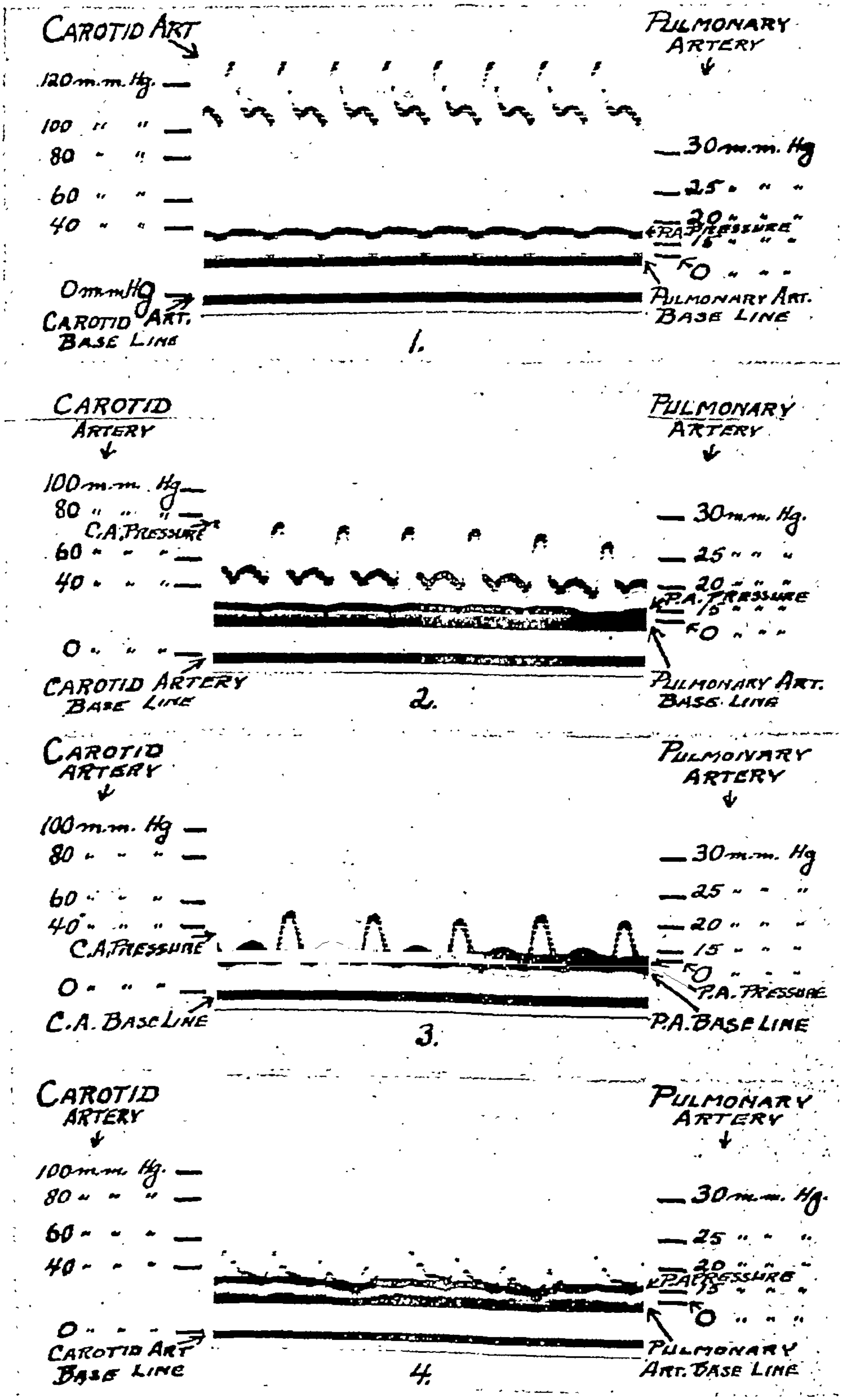

Fig. 3.—(*For legend see opposite page.*)

elevation of pressure could be demonstrated on the peripheral arterial side. The remaining portions of this paper will be devoted to the presentation of further evidence of a pathologic process within the lung resulting from acute peripheral arterial blood loss.

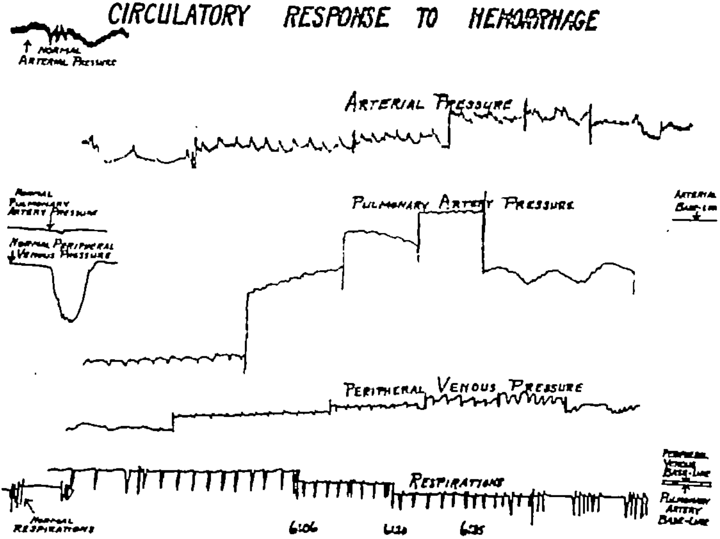

Fig. 4.—Kymographic record of pressures within the pulmonary artery, femoral artery and femoral vein taken before, during and following acute systemic hemorrhage.

The above is a photograph of a tracing of a 12-inch section from a kymogram taken during the period of greatest pressure rise in the pulmonary artery immediately following blood loss. The very delicate hairlike lines recording this pressure were not possible to bring out in a photograph of the original kymogram.

On the right will be found base lines of zero pressures. Normal tracings recorded before blood loss are shown on the left.

Hemorrhage was produced thirty-five minutes before the abrupt rise in pulmonary artery pressure. This is a typical response of pulmonary artery pressure to the congestion which takes place within the lung shortly after acute blood loss.

Note the elevation in the respiratory tracing and note its gradual fall at 6:20 and at 6:35 P.M. The lungs are held in hyperinspiration in an attempt to make available a greater number of alveoli for oxygen absorption. This curve returns to the normal level as the lung condition is cleared.

It seemed desirable to show by some quantitative procedure just when after hemorrhage one may expect pulmonary moisture change, for it was noted that histologic evidence of alveolar fluid accumulation depended upon the time which had elapsed following blood loss. This investigation seemed of value because it

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Fig. 3.—Pulmonary, artery pressure changes during and following hemorrhage recorded by Wiggers manometers. Optical record of systemic arterial (carotid) and pulmonary arterial tensions *1*, before hemorrhage; the standard deviations of the beam are indicated; *2*, during hemorrhage; note fall in both carotid and pulmonary arterial pressures; *3* following hemorrhage; note continued decline of both carotid and pulmonary arterial pressures; and *4* forty minutes following hemorrhage; note pulmonary artery pressure to be rising at this time, but carotid artery pressure remaining low.

was felt that information might be gained concerning some of the factors involved in the production of pulmonary edema which not infrequently complicates the course of a poor risk patient undergoing major operative surgery in which there has been considerable blood loss.

The plan of experimental procedure was to produce the circulatory changes of acute hemorrhage by instituting a fixed peripheral arterial blood loss in a series of dogs. The animals were sacrificed at varying intervals of time after hemorrhage, and the per cent of pulmonary moisture was determined using the seventy-two-hour desiccation and dehydration method of Elman. These findings were then compared with the per cent of pulmonary moisture found in dogs in which there had been no blood loss. Hemorrhage of 25 per cent of blood volume was used (equivalent to 1,300 c.c. in an average weight man), for it was believed that this might be comparable to average blood loss in severe traumatic and severe operative hemorrhage, and that it would give a good indication of the mechanism of pulmonary and circulatory change one could expect from a smaller blood volume loss.

METHOD

Healthy dogs were used. Nembutal was given intravenously as an anesthetic. Each animal was handled in exactly the same way, and after ten minutes of anesthesia, 25 per cent of the blood volume was removed free flowing from the femoral artery. At the end of the experimental period, each dog was killed by the injection of 10 c.c. of chloroform into the left chambers of the heart. In many of the experiments blood was taken for plasma protein and hematocrit levels, both before hemorrhage and before the sacrifice of the dog. Autopsy was performed immediately. Two specimens of lung, each of approximately 15 Gm., were quickly removed, blood was allowed to drip from the excised tissue for one minute, and each block was then dropped into a previously weighed glass stoppered bottle. Bottles containing wet lung were weighed, the stoppers then were removed, and bottles and stoppers were placed in a drying oven for forty-eight hours at 60° C. This was followed by further dehydration for twenty-four hours in anhydrous calcium chloride desiccation. At the end of the seventy-two-hour drying process, each bottle was reweighed and the per cent of moisture in each specimen was computed.

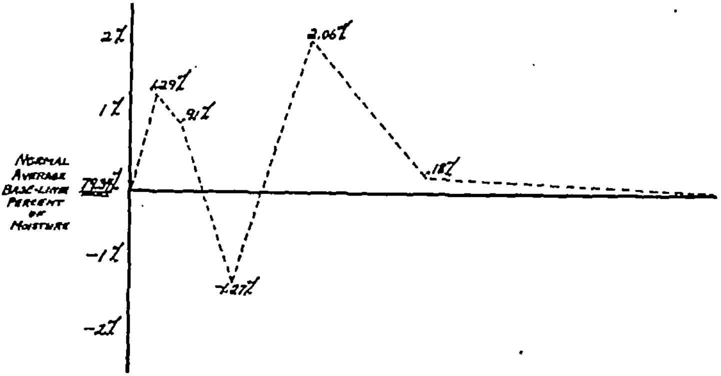
Seven groups of dogs were used in these experimental observations: Group A consisted of five controls sacrificed after ten minutes of anesthesia and without blood loss, and Groups

TABLE I. PULMONARY MOISTURE PERCENTAGE CHANGES FOLLOWING ACUTE ARTERIAL HEMORRHAGE

A	Five dogs, with no blood loss (controls)
B	Four dogs, sacrificed 10 minutes after hemorrhage
C	Five dogs, sacrificed 20 minutes after hemorrhage
D	Five dogs sacrificed 45 minutes after hemorrhage
E	Five dogs, sacrificed 1¼ hours after hemorrhage
F	Four dogs, sacrificed 2 hours after hemorrhage
G	Three dogs, sacrificed 4 hours after hemorrhage

Group	A	B	C	D	E	F	G
Pulmonary moisture percentages	79.98	80.72	79.89	78.12	80.53	79.39	79.60
	79.57	80.29	80.25	78.42	80.57	78.86	78.65
	78.93	80.88	79.78	78.14	80.83	80.20	79.77
	78.29	79.58	79.41	78.69	81.40	79.52	
	79.95		80.98	78.52	81.59		
Average per cent	79.34	80.37	80.06	78.35	80.98	79.49	79.34
Per cent change above or below the normal of Group A	79.34	1.29	0.91	–1.27	2.06	0.18	0.00

B, C, D, E, F, and G consisted of twenty-six dogs sacrificed ten minutes, twenty minutes, forty-five minutes, one and one-fourth hours, two hours, and four hours respectively after induced hemorrhage.



Graph 1.—Pulmonary moisture change following acute hemorrhage from the femoral artery (untreated), recorded in percentage above or below average normal.

RESULTS

In the Table I and Graph I an interesting pattern of pulmonary moisture change is noted during the first four hours following the controlled hemorrhage. An increase in the per cent of lung moisture is shown to have developed during the first ten to twenty minutes after an acute arterial blood loss. A fall below the normal level of pulmonary moisture was noted at the end of the forty-five-minute period. One and a quarter hours from the time of hemorrhage, the per cent of pulmonary moisture was again above the normal and there was a gradual leveling off to the normal base line during the subsequent four hours. It is admitted that the number of dogs involved in this experiment is small and inadequate when isolated statistically; considered, however, with the other experimental evidence of the paper, there seems to be a justifiable reason for submitting the data.*

SUMMARY

Hemorrhage of 25 per cent of blood volume from a peripheral artery produces a predictable pattern of moisture change within a four-hour period. Recent histopathologic studies indicate that pulmonary edema and hemorrhagic change may last several days. The quantitative changes in lung moisture of the first four hours are:

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*Recent studies suggest that this return to fluid equilibrium may not be permanent in all cases, and that the lung may again become edematous and that this lung pathology may not be so quickly removed. Autopsy has been performed in many dogs three to five days after a 25 per cent blood volume loss which had been followed by normal eating and drinking habits and normal activity with other dogs. Gross examination of lungs of some of these dogs has revealed patchy hemorrhagic areas 0.5 to 1.0 cm. in diameter on the pleural surface and on the cut surface. Microscopic study in these instances shows generalized edema, edema of the alveoli and alveolar walls, as well as capillary congestion and tissue hemorrhage. The point is raised that there may be but a temporary improvement in vascular imbalance and pulmonary congestion following peripheral hemorrhage and that pulmonary damage may be of clinical importance over a period of several days.

1. An initial rise in pulmonary moisture during the first twenty minutes following systemic hemorrhage.
2. A drop below normal levels at forty-five minutes.
3. A secondary rise in pulmonary moisture well above the normal averages by one and one-fourth hours.
4. A leveling off toward normal at two and four hours.

PART THREE

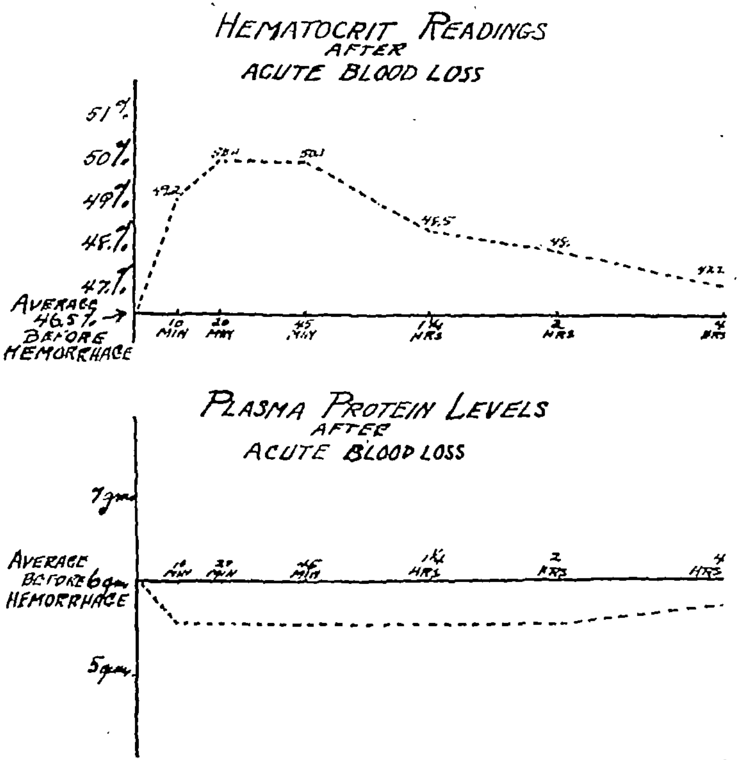
HEMATOCRIT AND PLASMA PROTEIN LEVELS FOLLOWING ACUTE BLOOD LOSS

In the plant and animal kingdoms normal life is maintained by fluid equilibrium within osmotic chambers. Plant life functions normally with a one-way directional flow, whereas in the animal world a complete circulation of fluid has been developed. Our circulatory system is a vast semipermeable osmotic chamber in which fluids and solutes pass back and forth between the tissue spaces and the blood vessels under varying systemic requirements. Two of the factors governing flow of fluid between the vascular system and the tissue spaces are: blood pressure change and variations in plasma protein concentration. It is of interest then, to note what changes in plasma protein concentration and hematocrit readings are to be found in acute hemorrhage. Table II and Graph 2 show the changes recorded in these experiments.

TABLE II. HEMATOCRIT AND PLASMA PROTEIN DETERMINATIONS FOLLOWING ACUTE BLOOD LOSS (FOUR DOGS USED)

TIME OF TAKING BLOOD	HEMATOCRIT (%)	PLASMA PROTEIN IN GM. PER 100 C.C.
Before hemorrhage	46.5	6.1
10 minutes after hemorrhage	49.2	5.6
20 minutes after hemorrhage	50.1	5.6
45 minutes after hemorrhage	50.1	5.6
1¼ hours after hemorrhage	48.5	5.6
2 hours after hemorrhage	48.0	5.6
4 hours after hemorrhage	47.2	5.6

During the first two hours following acute hemorrhage the plasma protein level is noted to be depressed, in grams per 100 c.c. of plasma, and the hematocrit reading to be elevated. This drop in plasma protein level and elevation of the hematocrit reading is due to the loss of albumin and liquid from the circulatory system to the tissues. In the pulmonary alveoli this alteration will subsequently be noted as a heavy albuminous precipitate (see Part Six). The hematocrit concentration is initially elevated at ten minutes, reaches its peak at forty-five minutes, and levels back almost to the normal or below by four hours. The observations on four dogs are here presented. They were used specifically for the purpose of determining the hematocrit and plasma protein levels at intervals of ten minutes, twenty minutes, forty-five minutes, one and one-fourth hours, two hours, and four hours after an induced hemorrhage of 25 per cent of blood volume. The figures here presented check quite identically with the hematocrit and plasma protein determinations made on dogs in each of the other experiments, but to incorporate all the data would be confusing to the reader.



Graph 2.—The plasma protein levels are depressed and the hematocrit readings are elevated during the first four hours following acute peripheral blood loss.

PART FOUR

QUANTITATIVE CHANGES IN PULMONARY MOISTURE FOLLOWING ACUTE SYSTEMIC HEMORRHAGE TREATED WITH INTRAVENOUS FLUIDS

The purpose of this portion of these experimental observations was to produce peripheral arterial blood loss, administer various intravenous fluids as therapy, and to ascertain what change in pulmonary moisture percentage might result. The reason for this investigation was the suspicion that the type, quantity, and method of administering postoperative intravenous fluids might contribute to the production of the pulmonary edema which occasionally complicates the course of the poor risk patient or the person who may be in good health yet is forced to undergo a major operative procedure with considerable blood loss. It was felt that these individuals were subject to such disturbance in circulatory and pulmonary physiology from the loss of peripheral blood that fluids given by the intravenous route might need to be given with caution for fear of impounding even greater pressures within the venous system against a damaged pulmonary endothelium and an embarrassed left heart.

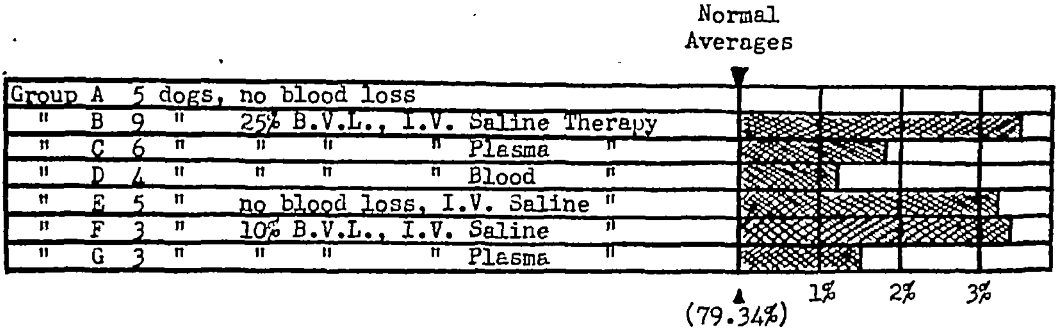


Fig. 5.—Pulmonary moisture percentage variations when various intravenous preparations are used after acute hemorrhage.

The plan of experimental procedure was similar to that of Part Two, except that various intravenous fluids were given as therapy for a four-hour period after hemorrhage. The circulatory changes resulting from acute hemorrhage were produced, as in Part Two, by instituting a 25 per cent blood volume loss; and, in addition, a smaller series was used in which a 10 per cent blood volume loss was sustained (500 c.c. in an average weight man). Physiological sodium chloride, plasma, and whole blood were the intravenous fluids given. When 25 per cent of the blood volume was removed, intravenous fluid was administered in the amount of two and one-half times this quantity (3,250 c.c. in an average weight man.) When 10 per cent of blood volume was removed, intravenous fluid was administered in the amount of four times this quantity (2,000 c.c. in an average weight man).*

METHOD

Healthy dogs were used. Nembutal was given intravenously as an anesthetic. Each animal was handled in exactly the same way, and after ten minutes of anesthesia the required blood volume was removed free flowing from the femoral artery. Intravenous fluids were started immediately and were given at the rate of forty drops per minute. Blood for plasma protein determinations and hematocrit levels was taken in selected cases before and after intravenous therapy. At the end of the experimental period, each dog was killed by the injection of 10 c.c. of chloroform into the left chambers of the heart. Autopsy was performed without delay. Two specimens of lungs, each of approximately 15 Gm., were removed, blood was allowed to drip from the excised tissue for one minute, and then each

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*The quantity of intravenous fluid to be given these experimental animals was determined by therapy used on the battlefields of World War II and in thoracic centers when major blood loss may be expected. During World War II it was common practice to give 3,000 to 6,000 c.c. and more of intravenous fluid to the casualty in profound hemorrhagic shock, even though in some instances pulmonary edema was observed to follow such treatment. In thoracic surgical centers it has become quite routine to give large quantities of intravenous fluid on the operating table and an equal amount in the immediate postoperative period. Therefore, it was decided to give these animals posthemorrhagic fluids which would be considered comparable to moderate amounts given the above-mentioned patients. Our purpose was to ascertain whether early pulmonary edema could be demonstrated by such treatment.

TABLE III. PULMONARY MOISTURE PERCENTAGES FOLLOWING SYSTEMIC HEMORRHAGE TREATED WITH INTRAVENOUS FLUIDS FOR FOUR HOURS (AUTOPSIED FOUR HOURS AFTER BLOOD LOSS)

A	Five dogs, no blood loss.
B	Nine dogs, 25% blood volume loss. Given intravenous saline
C	Six dogs, 25% blood volume loss. Given intravenous plasma
D	Four dogs, 25% blood volume loss. Given intravenous blood
E	Five dogs, no blood loss. Given intravenous saline
F	Three dogs, 10% blood volume loss. Given intravenous saline
G	Three dogs, 10% blood volume loss. Given intravenous plasma

Group	A	B		C	D	E	F	G
Pulmonary moisture percentages	79.78	82.92	82.25	80.99	80.81	80.30	81.98	80.73
	79.57	80.90	82.31	80.02	81.47	83.64	81.51	80.54
	78.93	82.22	80.97	80.59	79.93	80.12	82.46	80.50
	78.29	82.13	82.45	81.52	79.47	81.41		
	79.95	82.89		80.65		80.22		
				80.80				
Average per cent	79.34	82.13		80.74	80.41	81.15	81.98	80.61
Per cent change from A normal		3.50		1.86	1.18	3.28	3.82	1.57

TABLE IV. PLASMA PROTEIN AND HEMATOCRIT LEVELS FOLLOWING HEMORRHAGE AND VARIOUS INTRAVENOUS FLUIDS

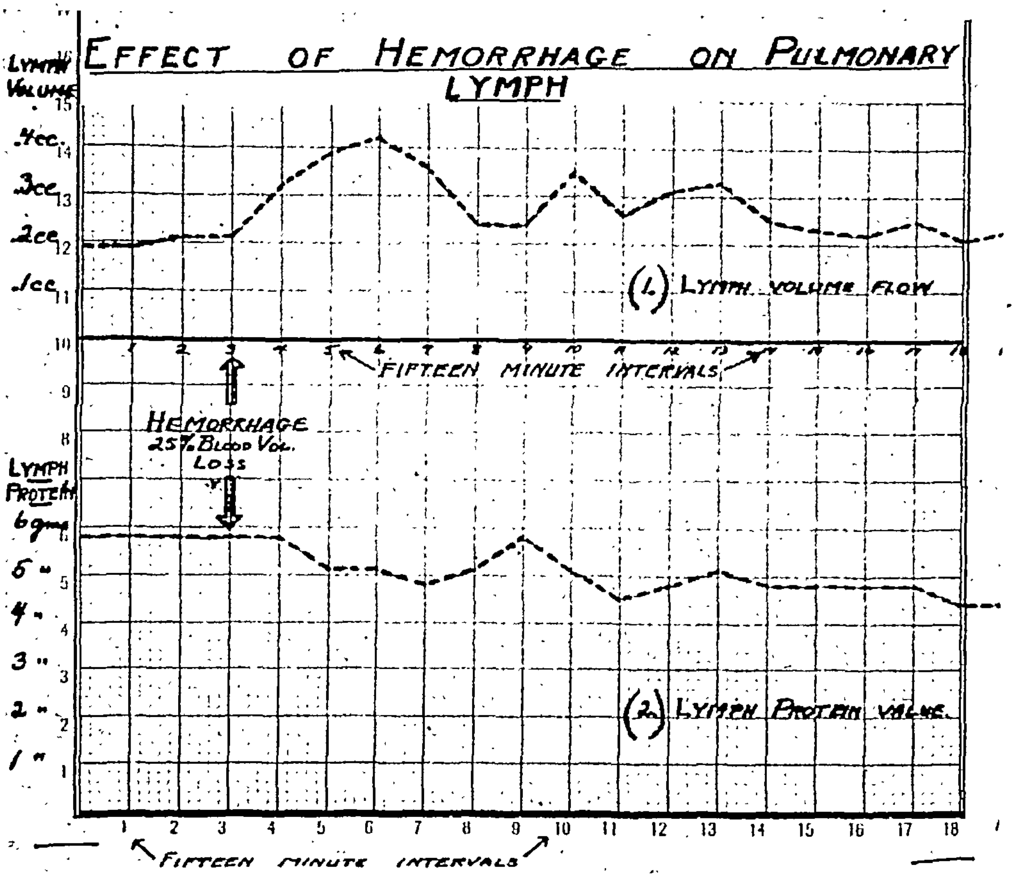
INTRAVENOUS FLUID	AVERAGE CHANGE IN PLASMA PROTEIN LEVEL (GM.)	AVERAGE CHANGE IN HEMATOCRIT LEVEL
25% blood volume loss, intravenous saline (5 dogs)	–1.4	–7.4
25% blood volume loss, intravenous blood (4 dogs)	–0.7	–0.2
25% blood volume loss, intravenous plasma (5 dogs)	–0.2	–13

block of tissue was dropped into a previously weighed glass stoppered bottle. The dehydration process was identical to that used in Part One. The per cent of moisture in each lung was computed and compared to the normals. The data are presented in Tables III and IV and Fig. 5.

RESULTS

From the above data and chart it will be noted that four hours after hemorrhage the greatest increase in pulmonary moisture occurred when physiologic sodium chloride was the intravenous agent, and that the greatest drop in plasma proteins resulted when this same fluid was used. When whole blood or plasma were the intravenous fluids administered, the per cent in pulmonary moisture increase was half that resulting from the use of intravenous physiologic sodium chloride. The quantitative findings in pulmonary moisture following hemorrhage seem to indicate that whole blood or plasma give comparable results as intravenous therapy, although even the use of these ideal fluids resulted in greater pulmonary moisture than is found four hours after untreated hemorrhage. Likewise it will be noted that following blood loss there was but slight alteration in the plasma protein level when whole blood or plasma were used as intravenous therapy, that there was considerable dilution of the plasma proteins when physiologic sodium chloride was given, and that the best hematocrit findings resulted when whole blood was used.

Group E was incorporated in this series in order to show that intravenous physiologic sodium chloride, alone, and without blood loss, will result in an increase in pulmonary moisture and a drop in both plasma protein and the hematocrit levels. In this regard it may be added that 2,000 c.c. of intravenous physiologic sodium chloride given to normal ward patients has resulted in a drop of 1.2 Gm. in the plasma protein level (unpublished).



Graph 3.—Pulmonary lymph is increased in quantity and depressed in protein content following hemorrhage.

SUMMARY

1. The administration of intravenous fluids after hemorrhage of 25 per cent of blood volume from a peripheral artery is associated with a greater increase in pulmonary moisture than is found four hours after untreated hemorrhage.

2. When physiologic sodium chloride is used the increase in pulmonary moisture is apparently twice that found when either whole blood or plasma are the intravenous fluids used.

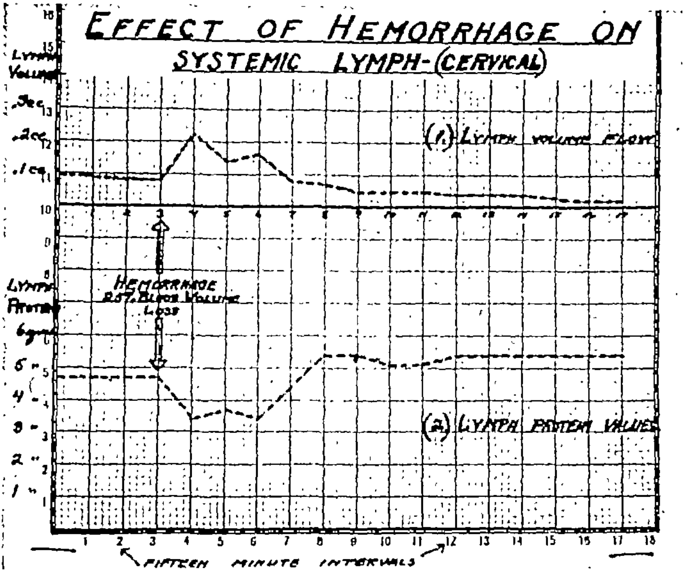
3. When whole blood is given intravenously following hemorrhage, the pulmonary moisture increase is slightly less than that resulting from the use of plasma.

4. When physiologic sodium chloride is given as intravenous therapy, there is a marked drop in plasma protein; whereas when plasma or whole blood are used this does not occur.

PART FIVE

CHANGES IN PULMONARY AND SYSTEMIC LYMPH FLOW FOLLOWING ACUTE PERIPHERAL BLOOD LOSS

In this next portion of the paper experimental observations are presented which indicate that acute hemorrhage from a peripheral artery has an immediate effect upon pulmonary lymph flow as well as systemic (cervical) lymph flow. When physiologic normal saline is administered intravenously following acute hemorrhage, it will be noted that an even greater stimulation of lymph flow from these areas is to be expected.

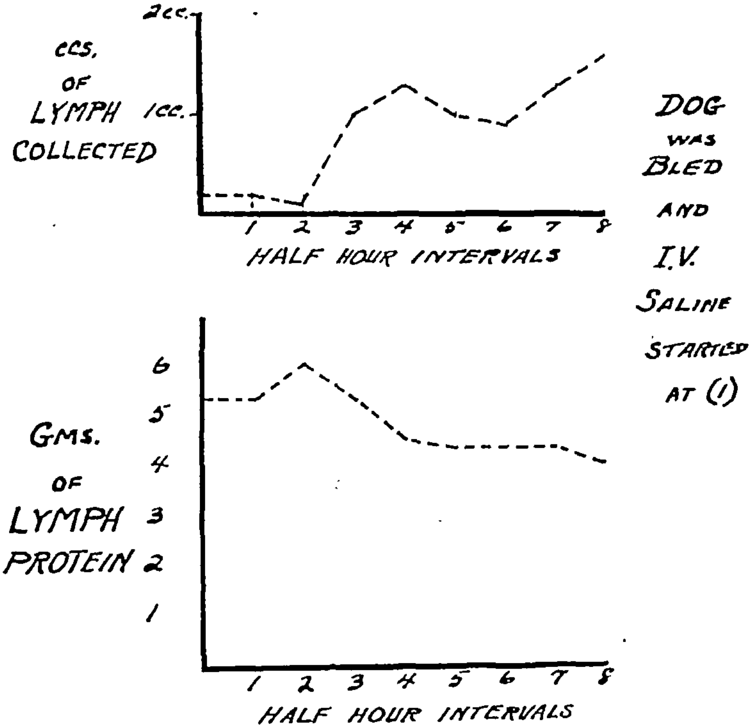


Graph 4.—Systemic lymph changes after acute hemorrhage.

METHOD

The plan of investigating alterations in pulmonary and systemic transudation was by quantitative and chemical lymph studies. Pulmonary lymph may be studied satisfactorily by canalization of the right pulmonary lymphatic duct (right thoracic duct) as it empties into the right subclavian vein, and systemic lymph flow may be studied by canalizing the cervical lymphatic ducts. Most of the lymph drainage of the right pulmonary lymphatic duct is of pulmonary origin and is for this reason a good indicator of the fluid economy of the lung.

The experimental procedure in each case was as follows: using large healthy dogs, and nembutal anesthesia, a cannula was inserted into the lymph duct (cervical or pulmonary, as the case may be) after the technique of Drinker. A normal lymph flow from the cannula was observed over the period of one-half to three-quarters of an hour at one-quarter hour intervals. Dogs were bled of 25 per cent of blood volume and the lymph flow was recorded both with and without intravenous therapy. Lymph protein determinations were made by the Van Slyke copper sulfate technique, a practical method of comparing the protein values of minute quantities of fluid. Each type of experiment was repeated several times. The data here presented are actual findings and seem to be typical responses for each type of experiment.



Graph 5.—Effect of hemorrhage and intravenous saline on cervical (systemic) lymph.

A. The response of pulmonary lymph flow to acute systemic hemorrhage is shown in Graph 3, in which the following may be noted: (a) a doubling in volume of lymph flow within forty-five minutes of acute hemorrhage followed by gradually leveling back to the normal; and (b) an initial drop in specific gravity and protein values of lymph noted in the first hour after hemorrhage.

B. The response of systemic (cervical) lymph flow to acute peripheral blood loss is shown in Graph 4 and the following points are to be noted: (a) an increase in volume of lymph flow during the first hour; and (b) a drop in specific gravity and lymph protein during the first hour.

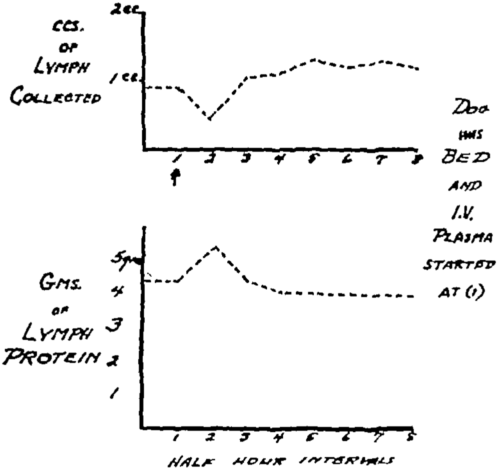
C. The effect of hemorrhage and intravenous physiologic sodium chloride on cervical lymph is shown in Graph 5. Of interest is: (a) a volume of lymph flow five to eight times normal; and (b) a drop in the specific gravity and lymph protein values.

D. The effect of hemorrhage and intravenous plasma on cervical lymph is shown in Graph 6. It is of interest to note: (a) the volume of lymph flow to be quite comparable to that of the hemorrhage experiment; and (b) but a slight change in specific gravity and of the lymph protein level.

PART SIX

PATHOLOGIC CHANGES WITHIN THE LUNG RESULTING FROM ACUTE ARTERIAL BLOOD LOSS

The observation that a pathologic state could be produced within the lungs by peripheral arterial blood loss was a finding of studies in which pulmonary lymph was being observed routinely when peripheral hemorrhage was induced experimentally (Part Five). At the time these lymph studies were carried out the experimental animals were observed over a period of four to five hours, then sacrificed, and immediate autopsy was performed. More attention was paid to the lungs than to any other organ because an attempt was being made to study the anatomic relations of the pulmonary lymphatics down along the mediastinum into the lungs.



Graph 6.—Effect of hemorrhage and intravenous plasma on cervical (systemic) lymph.

On opening the chest of many dogs, four hours after acute blood loss, the gross appearance of the pleural surface presented not the glistening pink of the normal lung but showed a mottled surface of dark red raised areas varying in size from that of a pin head to 0.5 cm. or more in diameter (Fig. 1). The cut surface of these lungs presented this same picture of patchy hemorrhagic infiltration. The areas of hemorrhage were confined more to the middle and lower lobes, with no apparent concentration of hemorrhage to any particular portion of a lobe, but with quite general distribution throughout the parenchyma. Blocks of such lung specimens were run through for histologic study and each presented the microscopic picture of alveolar and tissue edema, vascular congestion, frequent areas of compensatory alveolar overdistention and occasional hemorrhage. Fig. 6 is quite typical of these findings.

The desiccation experiments of Part Two were then conducted to prove the existence of an increase in the fluid content of the lungs when acute systemic hemorrhage was induced, and in each case histologic lung study was carried out. From Graph 1 (Part Two), showing pulmonary moisture change, Dr. Moore's Score Sheet (Table V), and Graphs 7 and 8 showing histologic changes in pulmonary edema and congestion, it became apparent that following acute blood loss there is developed a predictable cycle of pulmonary moisture change synchronized with comparable alterations in pulmonary lymph flow, pulmonary artery pressures, and histologic alterations within the lung.

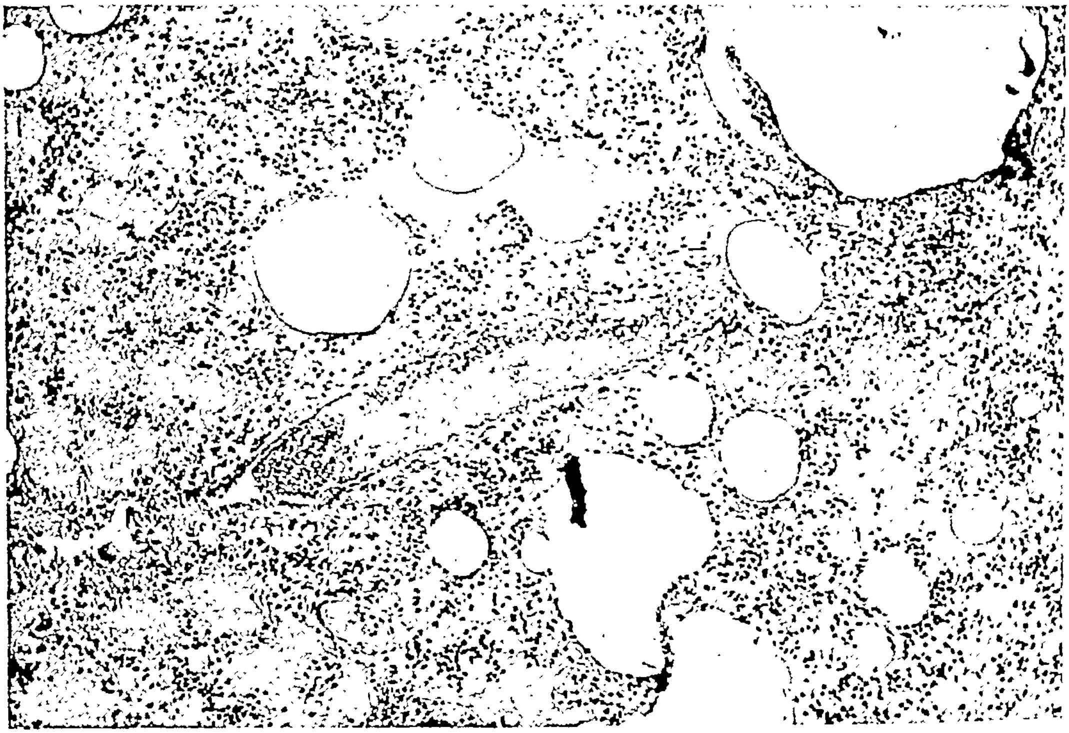
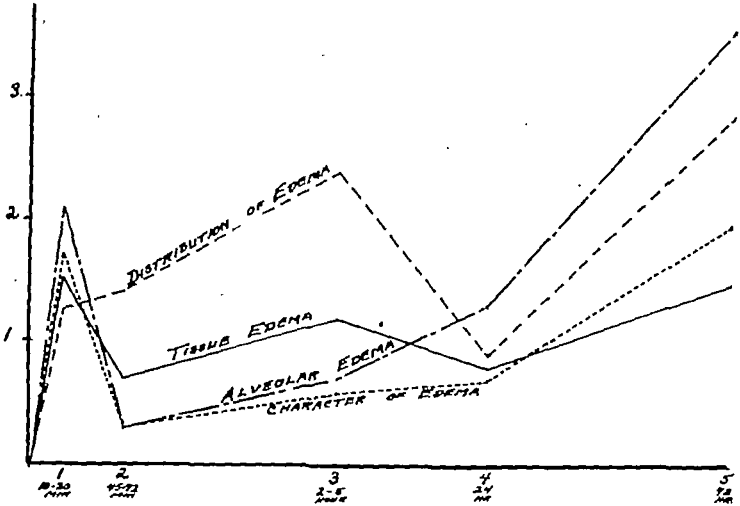


Fig. 6.—Photomicrograph of a dog lung four hours after acute loss of 25 per cent of blood volume from the femoral artery. Note alveolar edema and areas of alveolar over distention.

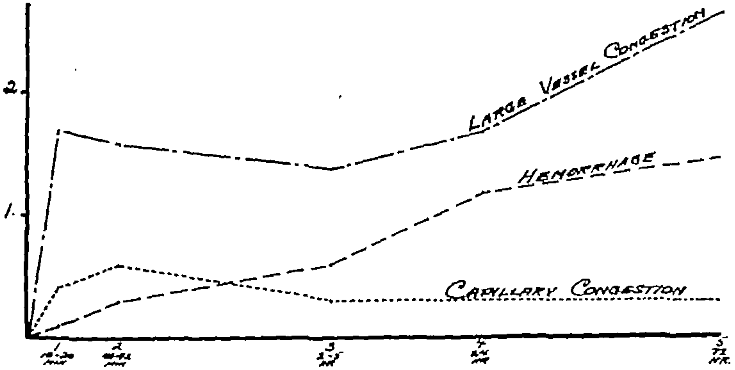
RESULTS

Tissue sections from the lungs of these experimental animals were studied by Dr. Robert Moore, Professor of Pathology in Washington University School of Medicine, St. Louis. Dr. Moore graded each lung section in categories pertaining to edema, hemorrhage, and vascular congestion, and adjudged them on a one to four basis depending upon the observed degree of pathologic change (Table V).

In order to arrive at a just basis of comparison of abnormal and normal, the graded tissue observations of four normal (unbled) dogs are presented in the first group; dog number 4 (Dec. 12) was sacrificed by injecting 10 c.c. of chloroform into the chambers of the heart without anesthesia, while the other three normals were sacrificed in the same manner, but after ten minutes of nembutal anesthesia. It will be noted that a grading of 0 was made only in



Graph 7.—Composite graphs of pulmonary edema following hemorrhage as to microscopic evidence of tissues, alveoli, distribution, and character of the edema in the lung. Data was derived from Dr. Moore's Score Sheet and curves were plotted according to distribution of the edema, the amount of tissue edema, the amount of alveolar edema, and the density of the edema. Points 1, 2, 3, 4, and 5 indicate the time interval after hemorrhage at which the dogs were sacrificed. The numbers on the vertical line refer to the units of pathologic change in Table V and represent increase over the normal.



Graph 8.—Composite graphs of pulmonary congestion following hemorrhage as to microscopic evidence of hemorrhage and congestion both capillary and large vessel. The time intervals at points 1 to 5 are the same as those in Graph 7. The numbers on the vertical line refer to the units of pathologic change in Table V and represent increase over the normal.

the categories of "alveolar edema," and "character of alveolar edema," and that there is a grade 2 capillary congestion in all four normals. However, using the same technique of killing the experimental hemorrhaged dogs, there is to be noted a very definite increase of graded value in each category of edema, hemorrhage, and congestion as compared to the findings of the normal.

There is an abrupt increase in alveolar edema at ten minutes following blood loss, a lesser degree of alveolar edema at twenty minutes, and a negligible amount at forty-five minutes; after this there is a definite trend to a reaccumulation of edema, reaching a peak at seventy-two hours, when this edematous condition is marked. It should be pointed out that the animals of the seventy-two-hour group were active in their dog pen after the removal of 25 per cent of blood volume and that they had normal eating and drinking habits.

TABLE V. DR. MOORE'S SCORE SHEET OF PATHOLOGIC LUNG CHANGES AFTER ACUTE HEMORRHAGE

TIME	DOG NUMBER AND DATE	EDEMA				CONGESTION		CHARACTER OF EDEMA
		GENERAL DISTRIBUTION	TISSUE	ALVEOLI	HEMORRHAGE	CAPILLARY	LARGE VEIN	
Normals	(4) Feb. 13	2	1	0	0	2	0	0
	(3) Feb. 13	1	1	0	0	2	0	0
	(4) Dec. 12	0	0	0	1	2	0	0
	(2) March 23	0	0	0	1	2	1	0
10 minutes	(1) May 9	3	1	4	1	2	3	3
	(2) May 9	0	2	0	0	2	0	0
	(1) March 30	4	3	2	0	2	3	1
	(1) May 15	3	2	3	1	3	1	2
	(2) May 15	2	3	2	1	4	1	1
20 minutes	(1) March 2	0	2	0	0	2	1	0
	(3) Feb. 27	1	2	2	1	2	2	3
	(4) Feb. 27	2	1	2	1	2	2	3
	(1) March 13	2	2	3	0	1	3	2
	(1) Feb. 27	3	2	3	1	4	4	2
45 minutes	(2) Feb. 13	1	1	0	1	3	0	0
	(1) Feb. 13	3	2	0	0	2	2	0
	(3) Feb. 9	2	2	1	1	2	2	1
	(3) March 16	2	1	0	1	1	3	0
	(2) March 1	2	2	0	1	2	2	0
60-75 minutes	(2) Jan. 31	1	1	2	1	2	1	2
	(2) Jan. 18	3	1	0	0	3	2	0
	(1) Jan. 31	2	1	0	1	4	4	0
	(1) Jan. 18	3	0	0	1	4	2	0
	(4) March 13	3	1	0	1	3	1	0
150 minutes	(2) Jan. 26	3	2	0	0	1	2	0
	(1) Jan. 26	3	1	0	0	1	1	0
240 minutes	(23) Jan. 16	2	1	0	1	3	1	0
	(1) March 20	4	3	2	3	3	3	1
	(1) March 23	4	2	3	1	3	3	2
300 minutes	(1) Feb. 16	3	1	0	0	3	0	0
	March 20	2	2	1	0	2	2	1
	March 23	3	2	2	1	0	2	1
24 hours	May 2	1	1	0	0	3	0	0
	(1) May 22	2	2	2	3	2	3	2
	(2) May 22	2	1	2	2	2	3	
72 hours	May 16	3	2	3	1	3	2	2
	Feb. 4	4	2	4	3	2	4	2
	May 4	4	2	4	2	2	3	2

PART SEVEN

DISCUSSION

From these observations it is conceivable that operative or traumatic blood loss can produce an imbalance in the circulatory system by the production of a pathologic state within the lungs. Such a change in normal pulmonary and circulatory physiology can have surgical importance, particularly as pertains to postoperative care and postoperative fluid therapy of critical or poor risk patients.

The physiologic changes which develop following acute blood loss are quickly produced. During the first ten minutes following a loss of 25 per cent of blood volume there is a marked increase in rate and depth of the respiratory effort (Fig. 7); a decrease in cardiac output; a fall of systemic, arterial, venous, and pulmonary artery pressures (Part One); a lengthening of circulation time (sodium cyanide test, unpublished); anoxia of circulating blood; an increase in endothelial permeability (Part Four); increase in pulmonary lymph flow (Part Five); congestion of the pulmonary capillary bed (Part Six); hemoconcentration (Part Three); a fall in plasma protein level (Part Three); and pulmonary edema as shown both microscopically (Part Six) and by desiccation experiments (Part Two).

Edema is a rapid development following acute blood loss, and is found in the lungs long before it is evident in other organs (unpublished observations). The pulmonary capillary system is affected by the same intravascular changes which are manifest elsewhere in the body, but in addition it is subject to intra-alveolar pressure changes and to back pressure from an incompetent left heart not adjusted to rapid vascular changes in blood volume.

The sucking effect of an increased intra-alveolar negative pressure is of great importance in a consideration of the pulmonary changes in question. Fig. 7 is a kymographic record illustrative of the immediate effect of simple peripheral arterial blood loss upon the respiratory cycle in the experimental animal. That this stimulation to respiration may be an important factor in the production of the initial pulmonary edema of the ten-minute period following blood loss is apparent when the following physiologic facts are considered.

In the normal resting individual a negative pressure of 3 mm. of mercury is exerted within the tracheobronchial tree with each inspiration, and this sucking effect is transmitted to the alveoli (Drinker).[4] Intratracheal pressure becomes zero at the end of inspiration and is followed by a slight positive pressure during expiration. Intratracheal negative pressures of 20, 30, 40, 50, and 70 mm. of mercury may readily result from tracheal obstruction (Drinker). Such negative pressures exceed the effective osmotic pressure within the capillary system and could draw transudates out into the alveoli even though no other factors were involved.

In 1933, Yamada[5] made pleural punctures on several hundred normal Japanese soldiers before and after vigorous exercise and obtained fluid in 20 per cent of these subjects at rest, and in 70 per cent after they had done severe work. In 1921 Graham,[6] using both the dog and human lung suspended in a bell jar, showed that expiration will produce pleural effusion by the squeezing effect of the respiratory effort, and that this effusion is increased when respirations are exaggerated. In 1943, Drinker[4] found that labored breathing produced an increase in pulmonary transudation in the dog as evidenced by increase in lymph flow from the pulmonary lymphatic duct; he has also shown that reduced oxygen supply increases pulmonary lymph flow, and he has pointed out that this increased transudation is due to an altering of capillary endothelium toward leakage as a result of a damaging effect of anoxia. Coughing, wheezing, choking, and production of tracheal phlegm are regularly observed when normal subjects hyperventilate themselves in competitive sports, running for trains, etc., and although these symptoms are usually transient, in some people a more lasting respiratory embarrassment may develop as a manifestation of acute pulmonary edema. If marked respiratory stimulation can produce systemic evidence of pleural effusion and pulmonary edema in the individual in whom there is normal cardiac reserve, then it should be possible to produce similar and even more severe changes in the experimental animal and in the postoperative patient as a result of the marked stimulation to respiration which results from the anoxia

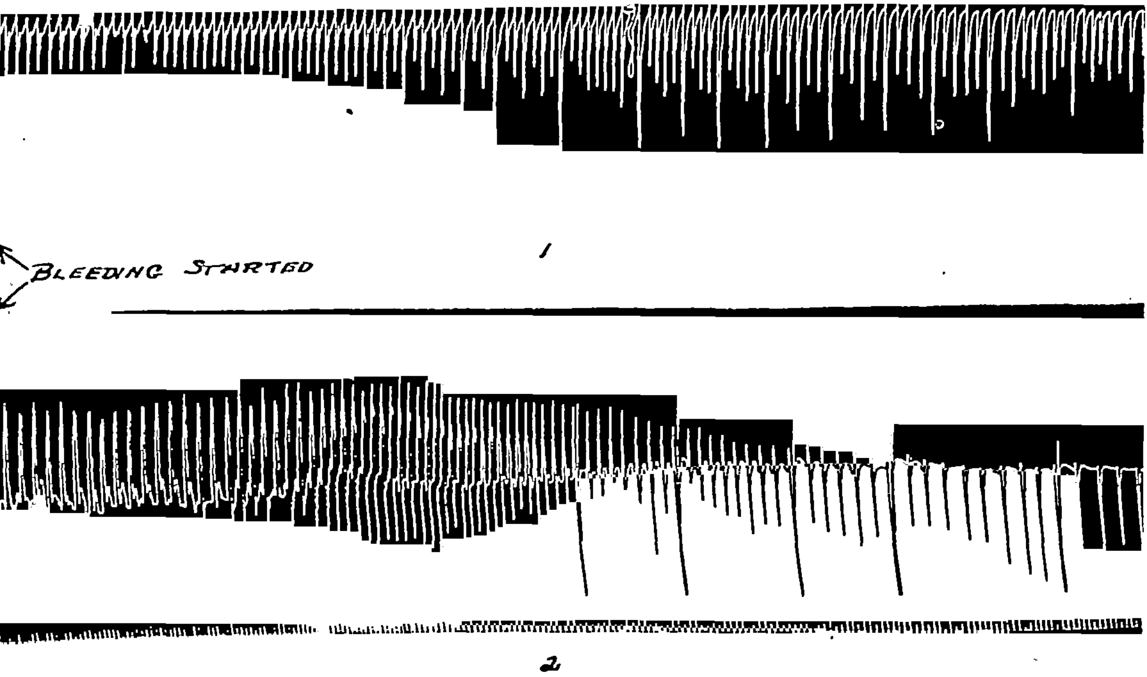


Fig. 7.—The effect of peripheral arterial blood loss on respiration. Kymograph of respiratory changes during acute hemorrhage.

Kymograph 1, with the tape about the mid-chest, demonstrates changes in intercostal breathing. Note period of hyperventilation followed by forceful breathing. As the period of apnea rises, (notch in upstroke) the chest is held in hyperinspiration.

Kymograph 2, with the tape about the upper abdomen, demonstrates changes in diaphragmatic breathing. Normal breathing is characterized by an upstroke in inspiration as the circumference of the abdomen decreases with the powerful sucking effect of chest muscles. Note the changes over to forceful diaphragmatic action pushing down upon abdominal contents and increasing the circumference of the abdomen, and note that a gradual elevation of the period of apnea is again shown as the diaphragmatic muscles also help to hold the lungs in hyperventilation (notch in upstroke).

of blood loss, for to this is added the factors of changes in endothelial permeability, alterations in the plasma protein level, and cardiac insult.

Forty-five minutes following acute arterial blood loss, a dehydration of the lungs is evidenced by a fall of pulmonary moisture percentage below the normal (Part Two), and there is noted to be histologic evidence of drying of the lung and overdistension of the alveoli (Part Five). Dehydration of the tissues of the body is a physiologic response to a loss of blood volume and will occur when intravascular osmotic pressures exceed hydrostatic pressures. At forty-five minutes there is an attempt to dehydrate the lung, intra-alveolar negative pressures are approaching normal, and even a blood of lowered osmotic pressure value will tend to draw fluid from the tissue spaces when hydrostatic pressures are low with blood loss (desiccation experiments of Part Two and the histologic observations of Part Six). However, pulmonary endothelium has suffered damaging effects during the first ten to twenty minutes following hemorrhage, fluid of high protein value has leaked into the alveoli, capillary congestion has developed, and frank hemorrhage has been produced within the lung. This attempt to dehydrate the lung is transient, the endothelial changes of the pulmonary capillary bed and the myocardial damage of the first twenty minutes have been too great. Pulmonary capillaries are still congested at forty-five minutes, the initial damage to this system is not quickly relieved and pulmonary edema is again produced by the drive of oncoming blood from the venous system plus back pressure from the left heart in congestive failure.

The pulmonary moisture elevation which is manifest one hour and a quarter after an acute arterial blood loss may have more than academic interest. High pulmonary artery pressures and peripheral venous pressures with low systemic arterial pressures have been demonstrated at this time interval, and the histologic evidence of alveolar edema again accummulating has been shown. In other words, endothelial damage has progressed now to the pathologic state, capillary pressures are elevating, and edema is again evident as endothelial permeability increases and as left heart inefficiency becomes more pronounced. Pulmonary edema and pulmonary congestion experimentally produced by mere peripheral blood loss may be the early manifestation of acute pulmonary edema which is seen following surgery. Histologic examination at this interval following blood loss shows pulmonary edema and capillary congestion (Part Five), and experimental observations have shown elevation of peripheral venous pressures as well as marked rise of pulmonary artery pressure (Part One), as pressure is impounded against damaged pulmonary endothelium and an incompetent left heart. As shown in Part One, this temporary pressure rise in the venous system is not associated with corresponding elevation in the peripheral arterial pressure following blood loss, and it seems probable that the vascular system is temporarily out of balance.

Pulmonary moisture seems to level back nearly to the normal expectancy four hours after hemorrhage and normal equilibrium is again established in the vascular system. However, recent observations indicate this to be a temporary adjustment and that many animals will show pulmonary edema and hemorrhage when sacrificed three to five days after blood loss even though there has been normal activity without symptoms of pulmonary disorder.

It is apparent that pulmonary obstruction to normal circulation is usually transient following a 25 per cent blood volume loss, and that in a matter of a few hours normal circulatory balance is re-established between the arterial and venous systems; however, it is also apparent that the pathologic changes which have taken place within the lung could not be corrected completely in this short space of time, and that this organ remains a potential source of circulatory obstruction should unwise intravenous therapy be given to tax the ability of an impaired pulmonary endothelium (as demonstrated in Part Four). When one considers the pulmonary moisture changes, the vascular imbalance (Fig. 2), the changes in pulmonary transudation, and the alterations in hematocrit and plasma protein levels which can be expected to follow acute arterial blood loss, a consideration of the possible dangers of type and quantity of intravenous therapy will seem necessary.

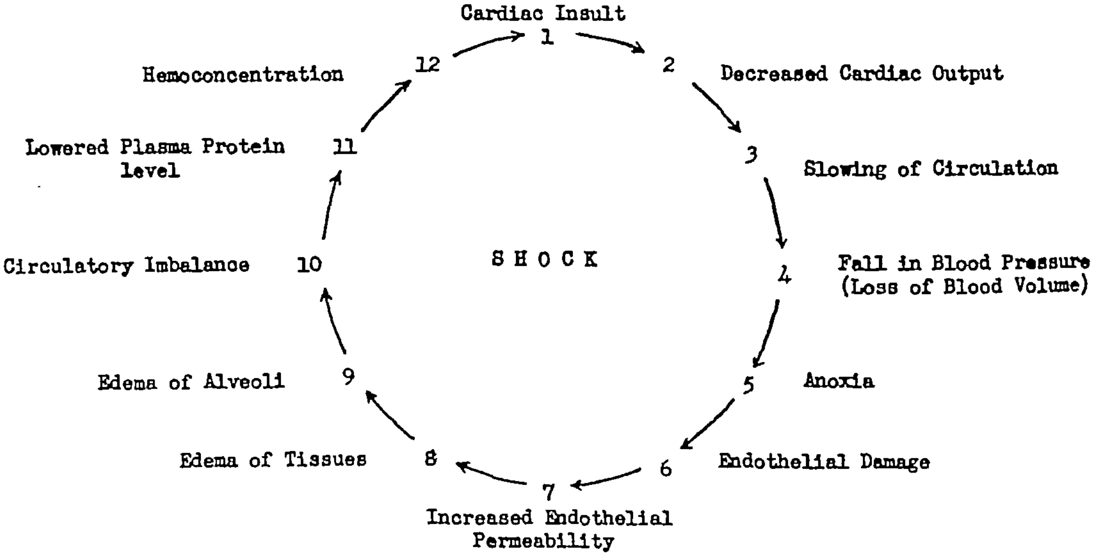


Fig. 8.—Schematic diagram of the possible mechanism of shock. It is probable that the shock state may be produced by a type of chain reaction initiated at any one of the above twelve points. Hemorrhagic shock emanates from point 4 with an actual loss of blood volume.

The entire vascular bed with its intricate ramification of capillaries is actually a vast osmotic chamber in which fluid passes back and forth between the interstitial tissues and the circulatory system. One of the important factors governing and maintaining normal fluid balance is the positive osmotic pressure effect of the circulating plasma proteins. The importance of maintaining normal plasma protein levels during postoperative care must be emphasized. When physiologic sodium chloride is used as intravenous therapy, there is a dilution of plasma proteins. Physiologic sodium chloride exerts no colloid osmotic pressure, and as an intravenous agent it dilutes the plasma proteins, colloid osmotic pressures decline, and fluid is allowed to leak into the alveoli and interstitial spaces from the circulatory system. Group E of the table serves to emphasize this point; in this group an increase in pulmonary moisture resulted from the administration of intravenous physiologic sodium chloride to unbled dogs.

On the other hand, it must be pointed out that the increase in pulmonary moisture after an intravenous therapy of either plasma or whole blood is considerably higher than is pulmonary moisture four hours after untreated hemorrhage. This brings up *the point that intravenous therapy following* severe hemorrhage will result in an aggravation of the pulmonary edema of blood loss, regardless of the type of fluid used. It must again be pointed out that peripheral blood loss produces a pulmonary block to normal circulation; and that if to this imbalance in pulmonary and circulatory physiology greater fluid volume be added to the venous side, then even greater pressures will be impounded against this abnormal pulmonary endothelium, and an aggravation of the pulmonary edema is the inevitable result.

To re-emphasize the remarks of the first part of this discussion, the colloid osmotic pressure of the blood passing through normal lungs is much higher than the capillary blood pressure, and this arrangement makes for a minimum amount of fluid in the alveoli. The osmotic pressure effect of plasma proteins causes absorption of interstitial moisture and holds fluid within the vascular bed, and this osmotic pressure is normally much greater than the hydrostatic pressure of blood tending to force fluid and solutes out of the capillaries. However, these are the conditions as they exist in normal fluid economy, before there is any blood pressure or osmotic pressure derangement, or endothelial damage to upset this delicate balance of the vascular system. Blood loss and intravenous therapy produce an imbalance in this fluid economy, and a pathologic state within the lung.

The inability of physiologic sodium chloride to exert positive osmotic pressure is just as apparent in humans as in the experimental animal. Its occasional use in the treatment of hemorrhagic shock attests to the unfortunate choice of this solution when plasma or blood are available. The ability of plasma to exert positive osmotic pressure is more apparent in the treatment of hemorrhagic shock than it ever could be in a laboratory experiment of this sort. The recognition of the life-saving virtue of this fluid has been one of the greatest medical accomplishments of World War II. The essence of plasma therapy is the ability of this fluid to maintain normal osmotic pressure within the vascular system. In many respects these experiments compare favorably with results in individuals who have been given in the one case physiologic sodium chloride and in the other case whole blood or plasma as intravenous therapy. Those receiving physiologic sodium chloride have a dilution of their plasma proteins, a loss of effective osmotic pressure, a *shift of fluid from the circulatory system* to the tissues, an accumulation of fluid within the alveoli, and a development of pulmonary edema. *When whole blood or plasma* are used as *intravenous agents*, this edema is kept at a minimum, provided rate and quantity of administration are carefully controlled *and venous pressures* are watched.

The reader must not construe from this paper that intravenous blood and plasma are condemned in the treatment of hemorrhage. These fluids, when administered in reasonable quantities, are absolutely necessary and are life-

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DISCUSSION

DR. I. A. BIGGER, Richmond, Va.—Dr. Eaton, it is my pleasure and privilege to present to you the Rose Lampert Graff Prize, awarded each year for excellence of work done, and presented at this Association's meeting. The committee this year felt that your work was of a very high order, and gives you this award as an indication of that feeling. We congratulate you and turn this over to you, and I am quite certain we will hear much more from you.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—I am delighted that the committee decided that Dr. Eaton should have this award. I want to tell you a little bit about this work, and how it was done, because I think there is a good object lesson in it.

Dr. Eaton, when in the Army, was assigned to duty in St. Louis, to be in charge of the Red Cross blood bank. This was an occupation which did not require all of his time. He began to come over to the Barnes Hospital and interest himself in various things, and he became very much interested on his own account in the amount of blood loss which was going on in connection with operations.

As a result of that, he has done this work, which I think is very important and very beautiful. He had no particular experience or training in physiological experimentation before undertaking this work.

After getting along to a certain point, it seemed desirable that he should learn to cannulate the right thoracic duct, and so it was suggested to him that it might be well to spend some time with Dr. Cecil Drinker.

The question then arose as to whether he could get away from his job at the blood bank in order to do this. It took only a little maneuvering in order to get him relieved from duty to go down and spend some time with Dr. Drinker, and when Dr. Eaton came back from this experience he was bubbling over with enthusiasm.

This is only a very small part of the very beautiful and magnificent job that he has done. He could have kept us here all afternoon telling us about the other things.

I think it is most significant that he has been able to explain, I think, in a very satisfactory way, the intimate connection between pulmonary edema and severe hemorrhage, no matter where the hemorrhage is from, whether it is from the lung or big toe or head, and, of course, having explained the pulmonary edema, it is easy enough to understand how to prevent it, and perhaps even to abort it when it is just beginning, because in the last analysis, it is a phenomenon of anoxia of the capillary endothelium in the lungs, and certainly, therefore, one should take immediate steps to improve the anoxia.

As he has already indicated, these steps are, first of all, the substitution of whole blood for that which has been lost. Under no circumstances would the admission of unbound water be allowed. I mean by that, that an electrolyte solution with no protein to bind the water should not be given. That is the worst thing that can be done. It is far better to do nothing than to give a patient unbound water under such circumstances.

Then, of course, one has the possibility of the direct administration of oxygen into the lungs, under pressure if necessary, and generally it is necessary in order to get the oxygen through the film of water that lines the alveoli.

DR. EATON.—I have nothing to add, except to express my appreciation to Dr. Graham for all he has done for me. He has done more for me medically than anyone in my experience.

PERICARDIAL COELOMIC CYST

LEON J. LEAHY, M.D., AND (BY INVITATION) GORDON J. CULVER, M.D.
BUFFALO, N. Y.

RECENTLY the opportunity to study a patient who had a relatively rare cystic lesion of the anterior mediastinum presented itself. The cyst was in close relationship with the pericardium and exhibited interesting radiographic, surgical, and pathologic findings. This case will be presented, and the origin of this and similar lesions will be discussed.

CASE REPORT

Mrs. G. I., aged 50, white, a housewife, was referred by: Dr. Leo A. Hellman, Port Allegheny, Pa.

Relevant History.—The patient was admitted to the hospital Jan. 20, 1946, complaining of hemoptysis, cough, and pain in her right lower chest. She had pneumonia with hemoptysis about twenty years previously. Six months following recovery there was another attack of hemoptysis which persisted over a one and one-half year period. Following this last attack the patient was well until July, 1945, when, after a rather exhausting month, she again began to have a cough with production of bloody sputum. These symptoms were present intermittently up until two weeks before admission. She had no hemoptysis in the two weeks prior to admission, but this reappeared five days after admission, which was the day following the initial bronchoscopy.

In December, 1945, the patient had a respiratory infection which was accompanied by severe headaches and rhinitis. This illness was followed by an increase in the severity and frequency of the coughing attacks with production of about one-half cup of clear to chunky sputum per day. Also following this episode she complained of pain medial to the tip of the right scapula. This pain was steady and aching in character, and sometimes sharply accentuated by deep breathing. During the four years previous to admission the patient experienced a weight loss from 140 to 115 pounds.

Physical Examination.—The temperature was 99.2° F. rectally, pulse was 80, respiration 20, and blood pressure 102/56. The trachea was in the midline. Respiratory movements were more marked in the upper than the lower chest, and on the left more than the right. There was dullness to percussion in the right costophrenic angle posteriorly, and at the level of the fifth rib anteriorly extending down to the costal arch.

No abnormalities of the heart were noted.

Roentgen Examination.—The chest showed a well-rounded, sharply demarcated, homogenous density on the right side, adjacent to the midline, blending with the cardiac shadow and touching the anterior chest wall. It did not appear to infiltrate the pulmonary parenchyma, but rather to be cystic in nature. Old pleuritic adhesions were noted at the peripheries of both hemidiaphragms. Radiographic studies of the oesophagus and stomach were done, and no abnormalities were demonstrated.

Laboratory Findings.—Urine: trace of albumin; leucocytes present on microscopic examination.

Blood: Red blood cell count, 4,300,000; hemoglobin, 11.9 Gm., white blood cell count, 6,600; differential count, normal; Wassermann and Kahn, negative.

Sputum: (concentration method) negative for acid-fast bacilli.

Read (by title) before Twenty-Sixth Annual Meeting of The American Association for Thoracic Suregery, Detroit, Mich., May 29, 30 and 31, 1946.

Bronchoscopy on Jan. 24, 1946, showed nothing more than chronic bronchitis, the type in which the membranes are very dry. There were numerous thin, yellow crusts in various places throughout the tracheobronchial tree. The patient coughed persistently during the bronchoscopy, but no blood was seen. Following withdrawal of the bronchoscope, the patient coughed and expectorated a small amount of bright red blood. An indirect laryngscopy was done immediately but no source of bleeding was found. The patient was rebronchoscoped at once. A small, red polyplike growth was found on the posterior wall of the left bronchus. This was biopsied. The sections showed normal mucous membrane.

The only positive finding on Jan. 30, 1946, was that the branch bronchus of the right lower lobe seemed to be partially occluded by swollen membrane and this bled freely when a forcep was introduced into the bronchus.

Correlating the clinical, roentgen, and bronchoscopic findings, it was felt that this patient had a benign tumor within the thorax. possibly a dermoid cyst or a hemartoma. It seemed most probable, considering the bronchoscopic findings, that the hemoptysis was the result of inflammatory bronchial disease and not related to the tumor per se.

Surgical Findings.—Under intratracheal anesthesia, an incision was made beneath the right breast anteriorly. Three inches of the fifth rib was resected with the costal cartilage. The lower and middle lobes were rather densely adherent to the pleura. It was necessary to remove the sixth costal cartilage before separation of the lower lobe from the mediastinum could be accomplished. This was done, and a cyst filled with clear fluid, which was about the size of a large orange, was carefully dissected and enucleated from the anterior mediastinum. This was removed intact. The attachments were ligated. These appeared to be fine fibrous bands containing a small amount of fat but no blood vessels of any appreciable size. The medial border of the cyst was in direct contact with the right margin of the pericardial sac. Laterally, there was a thin layer of areolar tissue separating the cyst from the mediastinal portion of the lower lobe.

The wound was closed in layers without drainage.

Postoperative Course.—The postoperative period was without incident. She was out of bed on the sixth day. Hemoptysis ceased on the seventh day. Follow-up examination on April 25, which was eighty days following surgery, revealed no complaints referable to her chest. There had been a weight gain and hemoptysis had not returned.

Pathology.—The specimen was a pear-shaped, cystic structure that weighed 145 Gm. and contained clear, watery fluid. The wall was very thin and somewhat idented along one border. There was very little fat tissue adherent to the cyst wall. Sections taken through the cyst showed a fairly clear picture of a simple cyst lined by endothelial cells. Most of the cells were flat but some showed a high cuboidal pattern. Morphologically these corresponded to mesothelial cells. No high columnar cells nor ciliated epithelium were seen. The cyst wall was edematous with a moderate inflammatory reaction. The mesenchymal portion consisted of distinctly edematous collagenous fibers. In some areas a few lymph follicles were seen with secondary germinal centers. The lining consisted of only one stratum.

DISCUSSION

In the literature, numerous cases of thin-walled cystic lesions of the anterior mediastinum have been reported. All of these have had one common feature, namely, their relationship to the pericardium.

A total of forty cases of pericardial diverticula were reviewed by Cushing[3] in 1937. In 1946, Mazer[9] reviewed thirteen additional cases and presented one more for a total of fifty-four. However, it was determined that thirty-eight, or 72 per cent, of this group were in reality encapsulated pericardial effusions (pseudodiverticula) most probably secondary to inflammatory lesions, thus being analagous to encapsulated empyema or effusion of the pleura. This leaves only sixteen cases of true diverticula of the pericardium. These have been called true "congenital diverticula."

Haas[6] has set forth the theory that these congenital diverticula form at a point of weakness in the parietal pericardium, where the fibrous layer passes out along the great vessels.

In addition to these pericardial diverticula, several cases of thin-walled cysts in close relationship to the pericardium have been reported.

In 1929, Dufour and Mourrut[4] presented the incidental autopsy finding of a thin-walled cyst arising from the interior of the parietal pericardium in a woman, 86 years old. There was no evidence of inflammation as a predisposing factor. Histologically, the wall appeared to be composed of flattened endothelial cells. This was interpreted to be a lymphatic cyst.

In 1934 Pickhardt[10] removed a thin-walled cyst from the left anterior chest of a 53-year-old woman. This cyst lay anteriorly in the chest extending from the pericardium to the costophrenic angle. The cyst wall showed a fibrous outer layer with an endothelial lining.

In 1937, a case of mediastinal cyst was presented by Churchill.[1] This patient showed, on roentgen examination, a mass at the right cardiac border slightly above the right auricle, which extended anteriorly to the chest wall. The mass was hemi-elliptical, being flattened against the heart. It was approximately 10 cm. in diameter. This was removed surgically. Pathologic studies showed an endothelial or mesothelial cyst and was called a "simple cyst" of the mediastinum.

In 1941 Curreri and Gale[2] reported a case of an anterior mediastinal cyst which was attached to the pericardium. This was a thin-walled cyst. No comment was included as to the histology of the wall. The author classified this as a "simple serous cyst."

In 1943, Greenfield, Steinberg, and Touroff[5] presented a case of a thin-walled anterior mediastinal cyst which had the same location as that presented by Churchill. The cyst was removed and histology of the wall showed a mesothelial lining. The cyst was thought to be analagous to that presented by Churchill and was called a "spring-water" cyst of the mediastinum.

In 1945 Kinsella[7] referred to these lesions as "pericardio-phrenic angle cysts" commenting on the subjective and objective characteristics.

From the above discussion it can be seen that there is considerable confusion in the literature concerning the true nature of cystic lesions associated with the pericardium, although on close inspection they appear to be very closely related.

In October, 1940, Lambert[8] presented two cases of thin-walled thoracic cysts and included another from Butler's clinic in an extremely comprehensive paper which serves to correlate all this conflicting data. He offered a simple embryologic explanation for what appears to be a somewhat complex problem. This explanation is based on the appearance of mesenchymal lacunae early in the life of the embryo which eventually coalesce to form the pericardial coelom. It is known that these lacunae are in no way fixed in their relationship to body segments. Connections between the pericardial coelom and the pleuroperitoneal coelom develop. Later, separation of the pleuroperitoneal coelom into two parts occurs. Lambert mentions various congenital anomalies which occur in failure of such normal developmental processes.

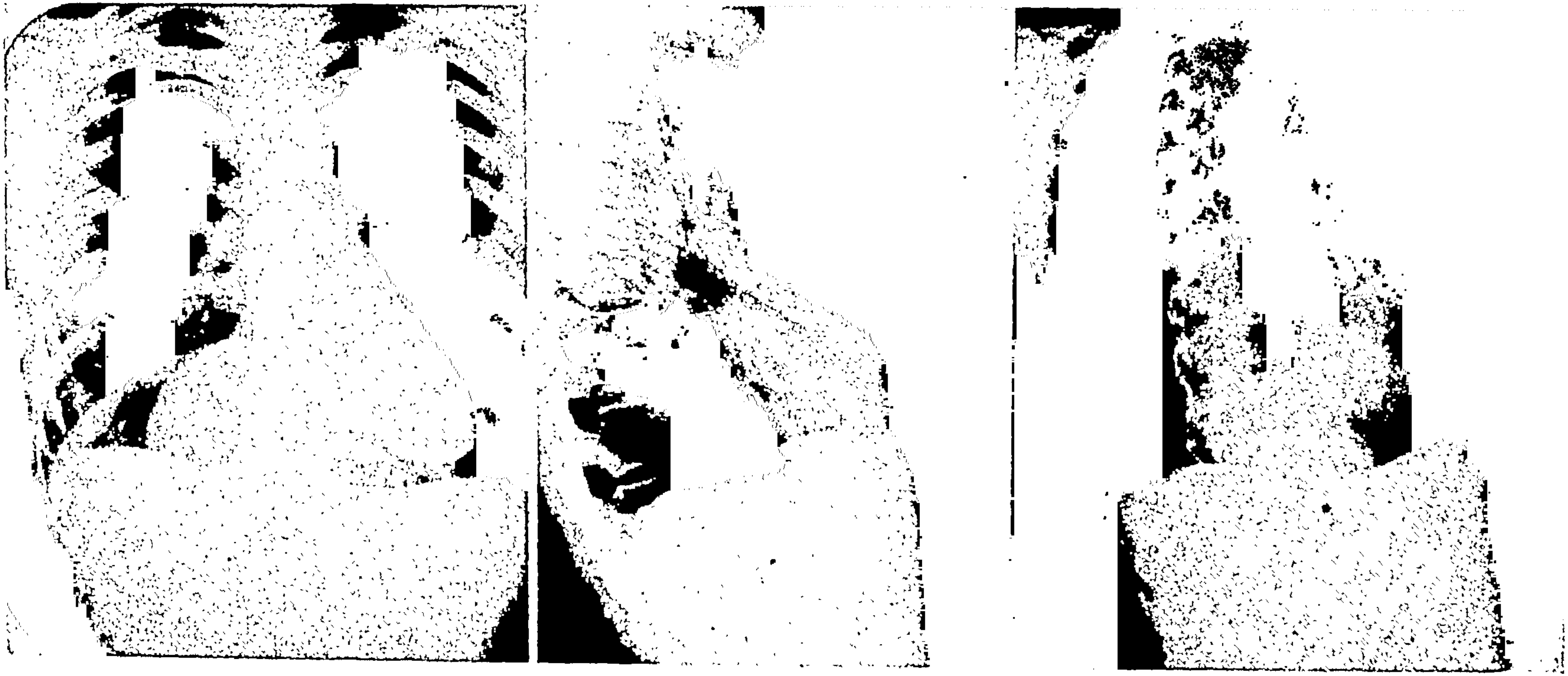

Fig. 1.—Films of the chest made in the posteroanterior, right lateral, and right anterior oblique projections to show the sharply demarcated mass lying just to the right of and adjacent to the heart shadow. This can be seen to lie against the anterior chest wall.

The important point to appreciate is the variations which may occur in the development of the lacunae which later form the pericardial coelom. These are listed by Lambert as follows:

1. Inequality in the developmental rate of *one of the primitive lacunae* which later form the pericardial coelom. This could easily explain the development of a true *congenital diverticulum.*

2. Failure of one of the primitive lacunae to merge with the others could well result in an independent cavity. That is, a cyst would be formed which could best be classified as a *pericardial coelomic cyst.*

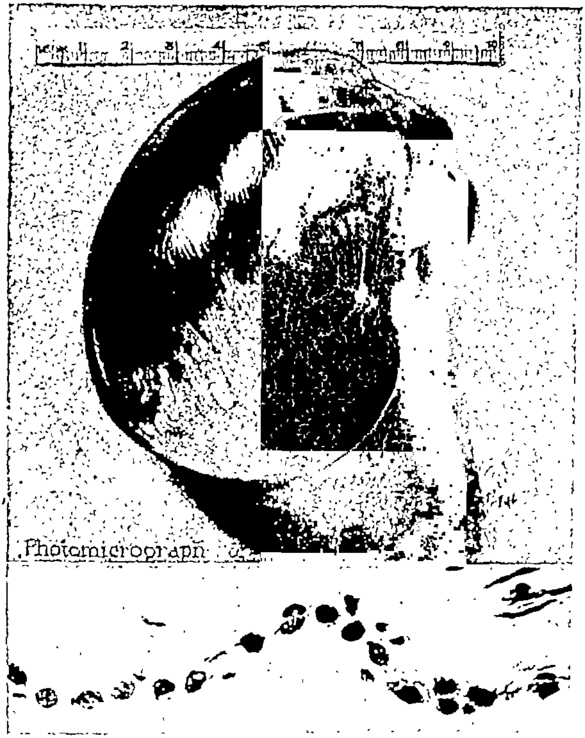

Fig. 2.—Drawing of intact specimen and photomicrograph of endothelial lining of the cyst wall.

Our thought is in accordance with Lambert. That is, we believe that all pericardial, or so-called "thin-walled" cysts of the anterior mediastinum in close association with the pericardium and "true pericardial diverticula," represent one and the same process. They are both developmental anomalies and not some obscure pathologic process to be identified as something different by each investigator who observes one of these relatively rare lesions.

For completeness, we must include a brief differential diagnosis of other cystic lesions which are found in the thorax. Echinococcus cysts exhibit a characteristic lining membrane. Embryonal cell-rest cysts from the respiratory and alimentary tracts show a cellular structure that readily identifies them. Dermoid cysts and teratomas likewise have a distinctive composition. Lymphagiomas represent a type of cystic tumor resulting from malformation of a group of lymph vessels, and are, therefore, of congenital origin. The histology of their walls is indistinguishable from that seen in pericardial coelomic cysts, so such an examination alone is insufficient for differential diagnosis. However, grossly lymphangiomas show multiple pockets with actual invasion of the surrounding tissues by the cystic tumor. The intimate association of these tumors with surrounding tissues makes enucleation at the time of removal quite difficult. These gross differences make it easy to distinguish between pericardial coelomic cysts and lymphangiomas.

It must be noted that the above differential study is entirely on a pathologic basis. The preoperative, clinical, and roentgen diagnosis is more difficult. However, the possibility of a pericardial coelomic cyst must be considered, when we discover a cystic tumor of the anterior mediastinum.

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THE REMOVAL OF FOREIGN BODIES FROM THE PERICARDIUM AND HEART

A MOVING PICTURE DEMONSTRATION

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COLORED moving pictures demonstrated the removal of a foreign body from an abscess in the pericardium and two operations for the removal of foreign bodies in chambers of the heart. These operations were taken from a series of operations in which 134 missiles were removed from the mediastinum. Of these foreign bodies, fifty-five were pericardial and thirteen were in the chambers of the heart. Seventeen cardiotomies were performed. There were no deaths.

Detailed discussion of the technique of cardiotomy for shell fragments in all four chambers of the heart is presented in a separate publication.[1] Further comments about the behavior of the heart during manipulation with electrocardiographic response to trauma, are also presented elsewhere.[2]

It was felt that certain foreign bodies should be removed from the heart: (1) to prevent embolus of the foreign body or associated thrombus; (2) to reduce the danger of bacterial endocarditis; (3) to avoid recurrent pericardial effusions; and (4) to reduce damage to the myocardium. Also, it is parenthetically added that pain and cardiac neurosis may be factors influencing the decision to remove some cardiac missiles.

It is emphasized that more foreign bodies, presumably within the heart, were allowed to remain unoperated than were removed. These were all asymptomatic.

It may be of interest to note that more than one-half of the patients referred to the thoracic center at the 160th General Hospital in the United States Army in the European Theater of Operations as having foreign bodies "in the heart" were found not to have these missiles in the heart when careful fluoroscopic examination was done. Furthermore, it was pointed out that approximately one-third of the missiles previously considered to be intracardiac, were not found in the heart at surgical exploration. These two factors make one feel that a foreign body cannot be conclusively diagnosed as intracardiac in position without surgical or autopsy exploration. In short, some of the cases of foreign body in the heart reported in the medical literature may not have been in the heart or its chambers. This would tend to make us feel that intracardiac foreign bodies are perhaps more hazardous than a straight statistical study of reported cases might indicate.

The experience represented in reports in the medical literature and in our own clinic clearly indicates that a large number, if not the majority of intra-

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cardiac missiles, may remain benign indefinitely. Conversely, our own observations and those of our colleagues lend clinical support to our view that some missiles should be removed for the outlined reasons.

OPERATION I

This operation was performed to remove a foreign body that could not be rotated out of the cardiac shadow on fluoroscopic examination. It was thought that it might lie in a herniation of the floor of the right ventricle. Anterior thoracotomy was effected through the left fourth intercostal space and extended transversely across the sternum. The pericardium was opened transversely. The exposed heart was kept moist with 1 per cent novocaine solution. When the pericardium was opened, the heart was small, firm, and readily contained within the pericardial sac. Exploration indicated that the missile lay in an abscess containing 18 c.c. of purulent material. (On culture it grew *Clostridium welchii.*) In order to expose the abscess and contained missile, the heart was lifted upward and forward out of the pericardial sac. This dislocation of the heart produced marked irregularity and cardiac dilatation. Electrocardiograms taken at the time in Lead II showed prolongation and slurring of the QRS complex interpreted as right bundle branch block. The P-R interval varied, and at one time the P wave disappeared as nodal rhythm supervened. Normal rhythm was resumed at the end of the maneuver. The pericardium was washed with saline after the missile had been removed and associated pus and debris had been excavated. The pericardium was closed with interrupted No. 0 chromic catgut sutures, leaving only a lateral window for decompression into the left pleural space to avoid tamponade. One hundred fifty thousand units of penicillin were injected into the pericardial sac through the decompression window.

The sternum was firmly reconstructed with silver wire and the ribs were approximated with pericostal sutures of double No. 0 chromic catgut. The muscles and fascia were then closed with interrupted No. 0 chromic catgut sutures and the skin edges were approximated with silk. The pleural space was not drained.

The patient made an uneventful recovery and the right bundle branch block cleared within forty-eight hours.

Discussion.—This sequence was presented to demonstrate: (1) the well-established fact that the heart does not tolerate dislocation from its position of optimum function. In this and other similar cases, it was found difficult to alter the position of the heart without interfering with its filling or emptying. (2) The value of firm reconstruction of the thoracic cage after cardiac exposure. This seems particularly important in firm sternal fixation as it reduces (a) postoperative shock and (b) postoperative bleeding. (3) The use of intercostal block with novocaine in oil before spreading the ribs. This reduces the shock of the operation and the postoperative discomfort of the patient. (4) The use of 1 per cent novocaine bath of the exposed heart to reduce cardiac irritability by (a) keeping the exposed heart moist and (b) by possible specific depressor action of the novocaine on the heart muscle.

OPERATION II

Cinefluoroscopic studies of a foreign body, presumably in the right ventricle, were shown. These were taken on one of our patients by Professor Barclay of the Nuffield Institute of Roentgenology at Oxford. These cinefluoroscopic studies were presented to illustrate the value of fluoroscopic studies in localizing foreign bodies that lay in or in relation to the heart.

This operation was presented to demonstrate the removal of a missile from the chamber of the right ventricle. The pericardium was exposed through a *left anterior fifth interspace incision. The fourth, fifth and sixth costal cartilages* were divided near the sternum. The left pleural space was freely opened in the usual fashion. The pericardium was opened transversely down to the left phrenic nerve. The missile in the right ventricle could be readily palpated. *The cardiotomy site was dictated by a zone of myocardial damage overlying the* contained shell fragment. (The zone of damage to the heart wall was produced by the massaging action of the muscle over the jagged metal.) Two staggered rows of hemostatic "U" sutures of No. 0 chromic catgut were placed about the damaged zone—the site for cardiotomy. Each suture was then tied loosely down to the myocardium to minimize the bleeding about the sites of suture perforation. The inner row of hemostatic sutures was then held in the assistant's hands. The epicardium was incised. The grasping forceps were then thrust into this chamber of the heart and the missile was grasped and teased around the papillary muscles and chordae tendineae to the heart wall. During this manipulation, it was possible to stabilize the missile and pinch off the apical portion of the right ventricle between the operator's left thumb on the anterior surface of the right ventricle and his left middle finger on the *diaphragmatic aspect of the heart. In this semi-isolated fashion, the grasping* forceps were maneuvered about in the chamber for more than three minutes. Forced transfusion of 1,000 c.c. of fresh whole blood was used to replace the loss of approximately 800 c.c. of blood from the heart. This blood replacement was effected during the manipulation and the succeeding fifteen minutes. As soon as the missile had been removed, the inner row of hemostatic sutures was crossed and tied. The outer row was then crossed and tied over a small free patch-graft of pericardium. A second, larger, free patch-graft of pericardium was tacked in place with interrupted No. 0 chromic catgut sutures. The anterior portion of the pericardium was then closed with interrupted sutures, leaving a decompression window 2 by 5 cm. in size in the lateral angle of the transverse pericardial opening. This allowed blood and effusion to escape into the left pleural space and safeguarded against the occurrence of cardiac tamponade.

The chest wall was then reconstructed in layers. Air was aspirated through a catheter as the left lung was expanded by the anesthetist. 100,000 units of penicillin were then injected into the pleural space through the catheter and the catheter was withdrawn. The pleural space was not drained. The patient made an uneventful recovery.

Discussion.—This sequence was presented to demonstrate: (1) Cinefluoroscopic studies of an intracardiac missile. (2) A technique of cardiotomy.

(3) Partial isolation of a portion of the right ventricle and open cardiotomy in that region for more than three minutes without fall of the systolic blood pressure below 110. This maintenance of effective blood pressure was due (a) to continued function of the remainder of the right ventricle and (b) to rapid blood replacement. (4) The importance of maneuvering the missile out through the forest of papillary muscles and chordae tendinea rather than by simple extraction at first blind grasp. (5) The use of free patch-grafts of pericardium over the incised zone in the right ventricular wall. (6) The preference for the use of wide, transpleural exposure for operations on the heart and adnexa.

OPERATION III

This was the third and successful cardiotomy for the removal of a missile that had migrated from the right ventricle to the right auricle at the first operation and from the right auricle *to the right ventricle at the second operation.* The technique of this intracardiac manipulation was essentially similar to operation II. The patient made an uneventful recovery.

Discussion.—This case was presented to demonstrate: (1) The use of the reverse Trendelenburg position and elevation of the patient's right side to prevent migration of the missile from the apex of the chamber of the right ventricle. (2) The use of an adherent pericardium to support sutures in the right ventricle. (3) Solid healing in a previous cardiotomy site that had been closed by No. 0 chromic catgut sutures and free pericardial patchgraft. (4) Damage to the myocardial wall by movement over an irregular fragment in the chamber of the heart. This damage was manifested by discoloration, flabby texture of the muscle, and paradoxical motion in the damaged zone.

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DISCUSSION

DR. HARRY R. DECKER, Pittsburgh, Pa.—This has been an amazing exposition of the technique and results of removing foreign bodies.

In my review of 100 cases in which missiles were removed from the heart, either the chambers or the muscle, prior to 1939, there was a mortality of about 20 per cent, and it was even a higher relative mortality in the cases in which the missiles were removed from the chambers.

I think that it can be said that at least half of the mortality resulting from the attempt to take foreign bodies out of the heart, of either wall or chambers, was the result of infection, setting up blood stream infection, or pericarditis, pneumonitis, or empyema.

We are better protected now, with the advent of the sulfa drugs and penicillin, in the prophylactic use against this very definite factor of mortality. Nevertheless, the success here has been due to dexterity and technical skill. Certainly the soldiers in this war are benefiting by a greater skill than did the soldiers in the last war.

The important thing is to decide which patients should be operated on and which should not.

The mortality statistics resulting in the last war showed that patients left with foreign bodies in the heart or in their chambers succumbed eventually in just about the same percentage as those that were operated upon. It would seem, therefore, that it was a standoff between conservative treatment and radical treatment.

Statistics do not mean very much. The last answer is that the case should be considered on an individual basis. Each case should be decided with reference to the patient's symptoms, interference with cardiac function, and neuropsychiatric reaction.

My interest in this subject was stimulated by a man who was incapacitated from economic inability to earn his living, because he had the knowledge of a foreign body which was really extrapericardial, although at the time we considered the case we thought it intrapericardial. When several different surgeons, including Frank Dolley, who saw the patient for me in California, finally convinced the man that the foreign body was in a location where it would never do any harm, he finally got back to a gainful occupation and was very happy about it.

We have patients who may need radical surgery to get them over their symptoms and get them back into ordinary gainful work.

DR. PAUL C. SAMSON, Oakland, Calif.—We have all been tremendously interested in this exposition of Dr. Harken's today and are thrilled to know that the risk of operation is certainly remarkably less than it has been in the past.

I believe and I am certain Dr. Harken agrees, that the advisability of removing foreign bodies cannot rest with a series of patients in which foreign bodies have been removed, until it is compared with similar patients in whom foreign bodies have been allowed to remain for a period of years and until patients of that type are evaluated very carefully as to complications. That, of course, must come with further study of cases which some of us may be able to garner from this war.

It might be interesting to compare the removal of foreign bodies in a base section center, which has been so ably demonstrated, with the removal of foreign bodies further forward in military operations.

At the end of the war, I had the opportunity of reviewing the surgery which was performed by surgeons of the Second Auxiliary Surgical Group, and a series of 75 heart and pericardial injuries were thus reviewed. This group formed 3.3 per cent of all the penetrating thoracic and thoracoabdominal wounds which members of the unit saw. In the series, 21 had foreign bodies, which were described in or about the heart and pericardium. It is of interest that 12 of these were not removed at the time of surgery, for one reason or another. Frequently removal was not undertaken because the patient's condition did not warrant further search.

Among the 21 cases there were eight deaths which occurred in forward hospitals, but on as careful a survey of the record as possible, it seemed that only two of the deaths were due directly to the foreign body itself. One of these to which I will refer later was from a missile embolic to the right ventricle, and the other was from a large fragment lying in a depression underneath the sternum. The deaths in the other six were not believed to be due to the presence of the foreign body.

We felt, therefore, that in all probability, unless the foreign body gave rise to definite symptoms during the first few days, when the patient was under observation at a forward hospital, it would probably be better to allow him to return to base section where further studies could be made and perhaps a leisurely approach to the removal of the foreign body would be possible.

One fatality was obviously due to an embolic foreign body. This patient was shot in the right flank by a shell fragment, which entered the cecum and disappeared somewhere in the midline. Laparotomy was done, the cecum repaired and the foreign body not found. Several days later he complained of chills and fever, and x-ray showed a definite shadow of a foreign body just below the diaphragm. He was believed to have malaria at one time, because of questionable positive smears, although it was never proved.

The patient seemed to recover, and on the tenth or eleventh day was evacuated and was dead on arrival at another hospital. Autopsy showed the missile lying in the right ventricle, having been washed up into the heart. The overlying myocardium for a distance of several centimeters was black, and death was due to the presence of the foreign body and the squeezing action of the myocardium on that foreign body.

One word, Dr. Beck, in connection with your speaking of the cardiac contusions; we had an interesting series of 26 cardiac contusions with and without laceration. These were proved to be contusions when the chest was opened for other reasons.

Contusions are very serious. There was a 40 per cent cardiac mortality, in this group. The patients stood operation very poorly, and I am sure that as time goes on, having seen so many of these contusions directly, we will be much more aware of them during civilian life.

DR. HENRY A. BRODKIN, Newark, N. J.—I would like to relate briefly a very unusual case not only pertinent to the paper under discussion but also apropos to the papers discussed on the subject of decortication of the lung. A similar principle is involved surgically and pathologically.

At the 15th General Hospital in Liege, Belgium, we received an officer who had had amputations of both legs as a result of an exploded land mine about one week before. The amputation stumps were encased in an adequate skin traction apparatus. The patient was anemic, dyspneic, apprehensive; and had an elevated temperature, small pulse, and low blood pressure. Veins of the neck were prominent and appeared distended. X-ray of the chest disclosed an enlarged cardiac shadow suggestive of a pericardial effusion with several small pieces of shrapnel superimposed on the cardiac shadow. The pericardial sac was aspirated and about 150 c.c. of a dark, brownish-red, bloody fluid was obtained. Following this there was a dramatic improvement in the patient, respiration and pulse improved and blood pressure rose to 110 systolic. Several days later he relapsed into his former condition and another pericardial tap was attempted and only about 10 c.c. of similar fluid was obtained. Cultures of fluids were negative. There was no improvement, and the patient's condition became progressively worse until he became semicomatose. Blood pressure fell to about 88/66, pulse was of small volume, veins of the neck quite prominent. Liver enlargement could not be definitely ascertained.

It was felt that we were dealing with a clotted hemopericardium that was contracting and interfering with cardiac diastole and had resulted in tamponade of the heart. In view of our experience with clotted hemothorax, it was felt surgically feasible to explore the pericardium and possibly relieve the compression of the heart. Under local anesthesia the pericardium was opened and the exact pathology was found here, as we saw in hemothorax. The pericardium was thickened, and soft fibrinous clot was removed with loculations of fluid. After incising the exudate over the heart, the encasing membrane could be peeled off with the finger in the same manner as it was done over the lung. When the heart was uncovered, the pericardium could be seen smooth and glistening and now the cardiac contractions, which had been comparatively slight, were strong and vigorous. The improvement in the pulse, pulse pressure, blood pressure was dramatic. An opening was made in the pericardial cavity into the pleural cavity so that any further oozing would go into the pleural cavity. The operative wound was closed without drainage. The improvement in the patient was rapid and dramatic. This was the first and only decortication of the heart for hemopericardium.

In addition I would like to mention briefly one patient who was evacuated into our hospital who had a left parasternal wound about the left third rib 2 inches long that was healed. He was sitting up perfectly comfortable in bed without any complaint. On looking at his x-ray there was a large metallic shadow partially within the cardiac silhouette and in the left hilar region. At first this was thought to be the soldier's aluminum identification tag. He had been evacuated from two other hospitals with similar pictures. It was not uncommon to see identification tags in the neighborhood of the upper mediastinum displayed in x-ray. Further x-rays, however, disclosed this shadow to be a large shell fragment approximately 2 inches long, about 1 inch wide and ½ inch in thickness weighing 100 Gm. This mass was removed under general anesthesia and found in a localized infected cavity resting on the convex surface of the pericardium and heart in the left anterior mediastinum. This patient made an uneventful recovery.

This case is mentioned because of the unusually large shell fragment resting on the heart and producing no symptoms.

DR. LAURENCE MISCALL, New York, N. Y.—In our center in Northern England, quite similar to the one in which Dr. Harken operated in Southern England, we did 440 thoracic cases in seven months. Of that number, 10 per cent were foreign bodies involving either the pericardium, the heart, or the great vessels.

The vast majority of these patients were referred to us by the medical services in the associated general hospitals for which we were responsible. They were admitted for several reasons. Some came for elective removal of an asymtomatic foreign body. These mainly involved the pericardium. However, we did see a fair number of foreign bodies in the wall or chambers of the heart and the pericardium. Many had been observed in other general hospitals for a period of time with apparent recovery. Some of these patients then had acute attacks with severe shock resembling coronary thrombosis or pericarditis.

We easily removed many such foreign bodies, with excellent results.

Here are a few slides which will illustrate why we removed some of them.

(Slide.) This is a first x-ray on a patient after he arrived at the thoracic center. He came for treatment of extensive empyema.

(Slide.) He had had decortication, and in none of the routine x-rays up to this time had this ventricular foreign body been demonstrated.

(Slide.) This happened to be foreign bodies in the anterior wall of the right ventricle. In removing, we found we had some difficulty because one of them did slip into the lumen of the heart. In removing these we placed a hammock of fine silk sutures directly under the foreign body. They traversed the cardiac lumen. They steadied the missile and heart during removal. The foreign body could then be attacked by sharp dissection. After visualization and removal, hemostasis was established easily by cross-tension on the hammock sutures. After the cardiac defect was closed by a few fine interrupted sutures, the hammock ones were cut close to the heart and pulled out. We found this more satisfactory than any other method.

(Slide.) This is the postoperative result.

(Slide.) This shell fragment proved to be in the right pulmonary artery. Just previous to this time, the right femoral region had been explored unsuccessfully for a foreign body. The patient complained of sudden pain in the right chest during the procedure. With this history and these plates we made diagnosis of pulmonary embolus (shell fragment from femoral region) which is visualized here.

(Slide.) We opened his chest and removed the foreign body from the right pulmonary artery. Part of the lower lobe which had sloughed was resected and the right middle lobe laid over it.

(Slide.) An arteriovenous fistula opened and caused this dilatation of the heart. We stretched the sphere of thoracic surgery a bit to interrupt the fistula with repair of both the artery and vein. These plates, taken on January 30, and February 10, show this rapid recovery.

(Slide.) This patient had an abdominal exploration for removal of this foreign body and suture of bowel. The foreign body was not found in the abdomen. He complained soon of precordial pain and was transferred to the center. On these plates and fluoroscopy we made a diagnosis of right ventricular foreign body.

(Slide.) It was removed from the right ventricle and here are his postoperative plates with apparent recovery.

(Slide.) This patient came to the thoracic center because of repeated hematemesis.

(Slide.) These plates showed the foreign body adjacent to a bronchus. Its tip was visualized through a scope on the left just below the upper lobe branch. It was not disturbed since we had seen one such removal followed by severe hemorrhage into the bronchial tree.

We explored the chest, (slide) mobilized the pulmonary artery, removed the foreign body and sutured the defects with excellent results. This patient went back to duty.

(Slide.) This patient with a foreign body in the superior vena cava had these almost normal x-ray plates taken about three weeks after injury. He had almost negligible hemothorax in his original plates.

(Slide.) This was removed easily by placing all sutures and controlling the circulation before extraction. Uneventful recovery followed. This is the postoperative plate (Slide).

Just what we accomplished in all of these cases is perhaps a question to be settled. I think we aided some of our patients, and especially those with embolic foreign bodies.

Of great importance is the demonstration that, with good anesthesia and trained teams, the elective removal of the foreign bodies of the heart can be an entirely safe procedure. We removed thirty-nine without any deaths.

It would be instructive to try to make a survey over the years of just what is the outcome after the removal of these foreign bodies.

DR. JEROME R. HEAD, Chicago, Ill.—Certainly one of the important differences between the recent war and World War I was the efficiency of the medical organization and the early and successful application to the ill and injured of modern medical and surgical measures. This was particularly true of thoracic surgery, which developed from terra incognita into a major and well-developed specialty between the two wars.

Because I was not active in this war, I hesitate to discuss the papers of the men who have been active and have done such excellent work, and do so only because I have been in a position to see and evaluate the late results of thoracic injuries and intrathoracic foreign bodies in the veterans of the first war. In a very large diagnostic, rating, and chest surgical service at Edward Hines, Junior, Hospital, I have seen all of the major thoracic problems which have developed in the veterans in the Mississippi Valley between 1926 and 1946. Certainly during the last war many foreign bodies were left in the chest and yet during that time we have seen only two cases in which they were causing trouble. Both of these patients had repeated severe hemoptysis. In both cases the foreign body was a rifle or machine-gun bullet.

PRESIDENT CLAUDE S. BECK, Cleveland, Ohio.—I should like to make one or two critical remarks about Dr. Harken's interesting demonstration.

In his first operation as shown in the motion picture the approach was to the left of the sternum and the foreign body lay to the right of the sternum. The sternum was cut across to get exposure. The sternum would not need to have been cut across if the approach had been made to the right of the sternum.

My second comment concerns the procedure made in removing the foreign body in the wall of the right ventricle. I believe the loss of blood could have been reduced by using a different technique, and would like to suggest in such a case the use of mattress sutures through the two walls of the ventricle so that the foreign body is exteriorized to some extent from the cavity of the heart. These mattress sutures can be held with some traction so that when the heart is opened the amount of bleeding can be controlled to some extent. This method has the additional advantage of fixing the foreign body in position so that it is not dislodged into the cavity of the heart during the manipulation of extraction.

DR. DWIGHT E. HARKEN.—On reviewing Dr. Decker's article, I was impressed with the fact that many of the foreign bodies that were said to be in the heart were so described on the basis of roentgenologic evidence alone. I am sure this will be Dr. Decker's recollection of various cases that he assembled from the literature.

It was interesting to note that in our own experience less than one-half of the foreign bodies sent to us as "in the heart" on x-ray evidence alone, were thought to be in the heart after fluoroscopic examination. It seems entirely likely that some of these cases of so-called "foreign body in the heart" might have reached the medical literature if we had not rotated the patients on fluoroscopy very carefully and found that they could be separated from the cardiac shadow.

Another factor is that, of those missiles that we thought might be in the heart, one-third at the time of surgical intervention were found to be, in fact, extracardiac in position. They might have been well within the shadow of the heart, but on exploration were found to lie high in the auricular-ventricular groove. These facts tend to make gross statistics derived from all cases reported in the literature, falsely optimistic.

In short, I am sure Dr. Decker will agree that one should not accept the statistics on foreign bodies in the heart unless they have been established as in the heart by operation or autopsy.

I was pleased to hear him discuss the psychological reaction of the patient. During the course of the evolution of our rationale, I sought the best counsel available. Being in England, one of the men was Professor Gray Turner. He said, "In addition to the factors that you mention, young fellow, a great deal will also depend upon the individual's reaction to the knowledge that he harbors an unwelcome visitor in one of the citadels of his well-being."

I particularly appreciated Dr. Samson's comments and the addition of a case that does support our rationale for the removal of some of these cardiac foreign bodies. Dr. Samson has said privately, he is sure that this patient could have been salvaged by the removal of the foreign body.

I emphasize again the fact that we elected to leave more than we removed. It has been quite clear that we are not sure of the ultimate fate of these people, but at least they are now all clinically well, and there were no surgical deaths.

Dr. Miscall added very interesting embolic cases. I have removed three embolic shell fragments from intrathoracic blood vessels, two to the pulmonary artery and one to the innominate artery. All patients did very well.

As to Dr. Beck's comment about the exposure of the first foreign body, I am sure that he is right. We might have done better to use a sternal dividing incision or even a similar approach to the one used, but an interspace lower and on the right side, and attained much readier access to the foreign body. That case was presented to demonstrate the point about dislocation of the heart (that admittedly was forced upon me by the use of an improper incision). The production of right bundle branch block is significant.

Dr. Beck's second suggestion, namely, a sling suture might be used for fixation of the missile before opening the ventricle, is well taken. This technique was never used, however, for fear that the missile or associated thrombus might be dislocated in the maneuver before I gained control of the fragment by the open technique. This missile was the only one that got away.

I hope we have learned more from this than that we can remove foreign bodies from the heart, and that we have added to our understanding of the behavior of the heart during manipulation.

THE USE OF FLUORESCEIN TO DEMONSTRATE THE EFFECT OF ARTIFICIAL RESPIRATION UPON THE CIRCULATION

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New York, N. Y.

UNTIL recently, no satisfactory method was known for demonstrating the effect of artificial respiration upon the circulation of the blood alone. This effect can now be observed by the use of tracer substances injected into the vascular system of animals. This is done immediately after death from asphyxia when there is no active heartbeat. The tracer substances which we have used are: radioactive sodium,[1] oxygen,[2] and fluorescein. This report describes the use of fluorescein only.

The purpose of this study was to determine the effect of inflation and deflation of the lungs upon the circulation, using a tracer substance, fluorescein, which is grossly visible with the aid of an ultraviolet light. Also to observe the extent of any movement of the blood column produced by such pulmonary inflation and deflation.

Nineteen experiments were performed on dogs.

TECHNIQUE

A medium-sized dog was anesthetized by the intraperitoneal injection of pentothal sodium. An endotracheal tube with occlusion cuff was inserted. The occlusion cuff was inflated to make the tube leakproof and to provide a direct communication with the lungs. The femoral artery and vein on one side were exposed, and 20 to 40 mg. of heparin were injected into the vein. Approximately ten minutes later, the endotracheal tube was completely clamped off. The dog succumbed to obstructive asphyxia in about eight to ten minutes. Following the cessation of respiration and cardiac function, the tube was left clamped for an additional twenty to thirty minutes to insure no subsequent heart activity. In many of the animals, electrocardiograms were taken to demonstrate that the heart had completely ceased to function. After this time, the animal was placed on an inclined plane with the head elevated about 10 degrees and 2 c.c. of a solution of fluorescein was injected into the femoral vein or artery. The endotracheal tube was unclamped and connected to a resuscitator.

The resuscitators which were used delivered either positive pressure only or negative pressure only, or positive pressure alternating with negative pressure. The pressures were a maximum of plus 14 mm. of mercury and minus 9 mm. of mercury.* The rate was sixteen to twenty per minute.

The inflation and deflation of the lungs was continued for approximately one hour, during which time frequent observations were made with an ultraviolet light for evidence of fluorescein in the skin or mucous membranes of the oral cavity.

Fluorescein is a substance having the property of emitting a very intense golden-green light when it is illuminated by ultraviolet light of approximately 3,600 angstrom units.

From the Thoracic Surgical Service of the New York Medical College, Flower and Fifth Avenue Hospitals, and the Metropolitan Hospital.

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*The resuscitators were built by the J. H. Emerson Company.

This is just at the borderline of the visible light. Fluorescein seems to be nontoxic and to have no effect whatsoever on the vascular system. It has a small molecule (molecular weight, 328) and therefore diffuses rapidly into the interstitial spaces if and when a filtration pressure is present in the vascular system. A 5 per cent solution of fluorescein to which was added 5 per cent of sodium bicarbonate was used.* Under normal circumstances, it is rapidly excreted into the urine. A small, inexpensive mercury vapor bulb covered by a double layer of purple Corning Glass No. 387 was used as a light source† and after the injection of the fluorescein solution, the entire body surface of the animal and the oronasal mucous membranes were examined at short intervals for the first typical luminous appearance of the dye. The intravascular injection of fluorescein has many applications in the determination of circulatory sufficiency in different areas of the body and has been described in detail elsewhere.[3-5]

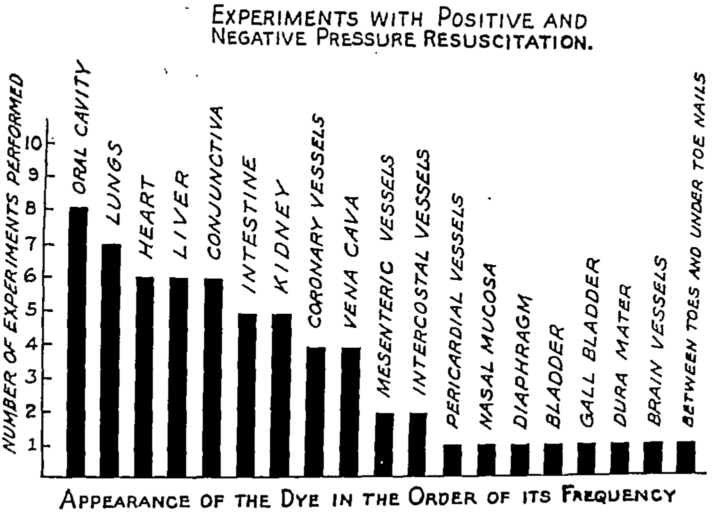

Chart 1.—Appearance of the dye at various sites when alternating positive and negative pressures are used.

The types of resuscitation used were alternating positive and negative pressure, positive pressure only, and negative pressure only.

In nine experiments, alternating positive and negative pressure resuscitation was used. The fluorescein was injected into the femoral vein in six, into the femoral artery in three experiments. The time elapsed between the beginning of resuscitation and appearance of dye in the oral cavity or conjunctivae varied from ten to thirty-seven minutes.

Autopsies were performed on nine dogs after twenty-two to eighty-one minutes of resuscitation and the presence of the dye in the various parts of the body was noted. In the order of frequency, it was observed in the following sites. (See Chart 1.) The numbers after the organs listed refer to the number of times the substance was found: oral cavity (8), lungs (7), heart (6), liver (6), conjunctivae (6), intestine (5), coronary vessels (4), vena cava (4), mesenteric vessel (2), intercostal vessels (2), pericardial vessels (1), nasal mucosa (1), diaphragm (1), bladder (1), gall bladder (1), dura mater (1), brain vessels (1), between the toes and under the toenail (1).

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*Supplied in ampules by C. F. Kirk & Co.

†Supplied by George W. Gates and Co.

Complete autopsies were not made in all animals and that fact accounts for the appearance of the dye reported in the oral cavity and not reported in the other organs. The brain was autopsied in only one animal.

Two experiments were done with negative pressure only. In both occasions the dye was injected into the femoral vein and autopsies performed in forty-five and forty-four minutes, respectively, after the beginning of resuscitation. The dye was found to be limited mostly to the venous side of the vascular system. At autopsies the dye was found in the following sites in order of its frequency (see Chart 2): vena cava (2), lower abdominal wall (2), heart (1), lungs (1), liver (1), gall bladder (1), sigmoid colon (1).

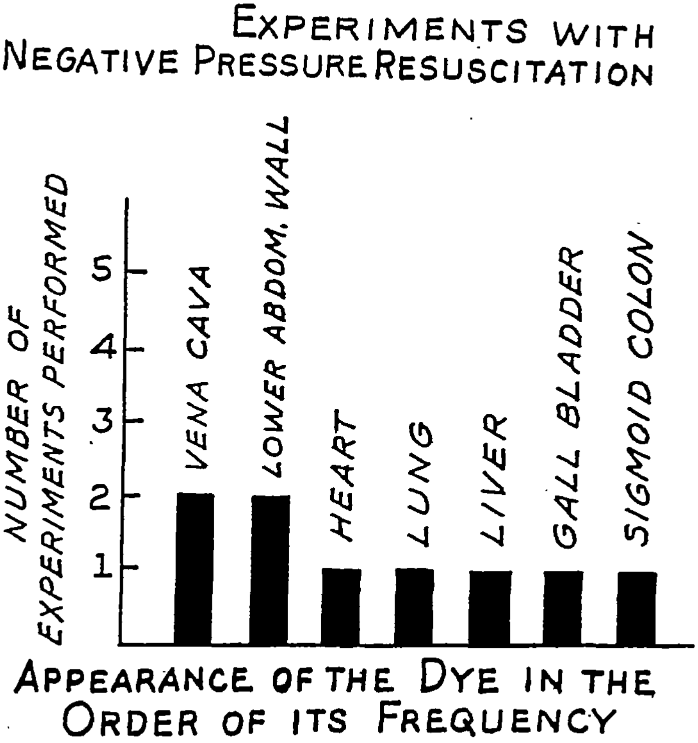


Chart 2.—Appearance of the dye at various sites when negative pressure only (suction) was used.

Four experiments were done with positive pressure only. The dye was injected into the femoral vein in two experiments and into the femoral artery in two experiments. The fluorescein was first observed within the oral cavity after sixteen to thirty minutes following the beginning of resuscitation. Autopsies were performed after twenty-nine to forty-six minutes of resuscitation and fluorescein was found in the following sites in order of its frequency (see Chart 3): vena cava (4), oral cavity (2), liver (2), kidney (2), lower abdominal wall (2), conjunctivae (1), axilla (1), lungs (1), gall bladder (1).

Four control experiments were performed. In one, fluorescein and heparin were used as in the other experiments but no resuscitation procedure was used and the dog was kept in a horizontal position. There was evidence of some local and downward diffusion of the fluorescein. In one, the diffusion was of a local nature, since this dog was in Fowler's position during the experiment. Two experiments were done without the injection of heparin. In both occasions, clotting of the blood prevented the movement of the blood within the vascular bed in spite of the resuscitation procedures.

DISCUSSION

These experiments demonstrate that the inflation and deflation of the lungs produces a movement of the blood in the vascular system. By the use of a tracer substance this movement is seen to be in the normal direction from vein to right heart, to lung, to left heart, to artery, and back to vein again, thus making a complete circuit. By these experiments we have demonstrated the possibility of circulating the blood over the entire body without the benefit of any heart action but by the simple inflation and deflation of the lungs.

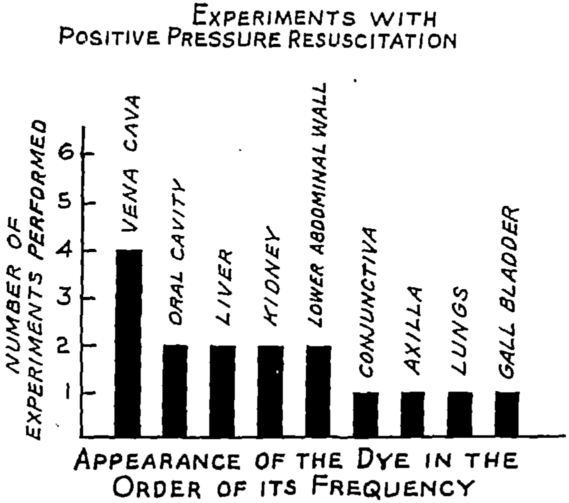


Chart 3.—Appearance of the dye at various sites when positive pressure only was used.

The brain was examined in only one experiment, and this autopsy showed that the brain vessels as well as the brain substance contained large amounts of fluorescein.

To prevent the effect of gravity on the diffusion of the dye, the animals were placed on an inclined plane with the head elevated approximately 10 degrees.

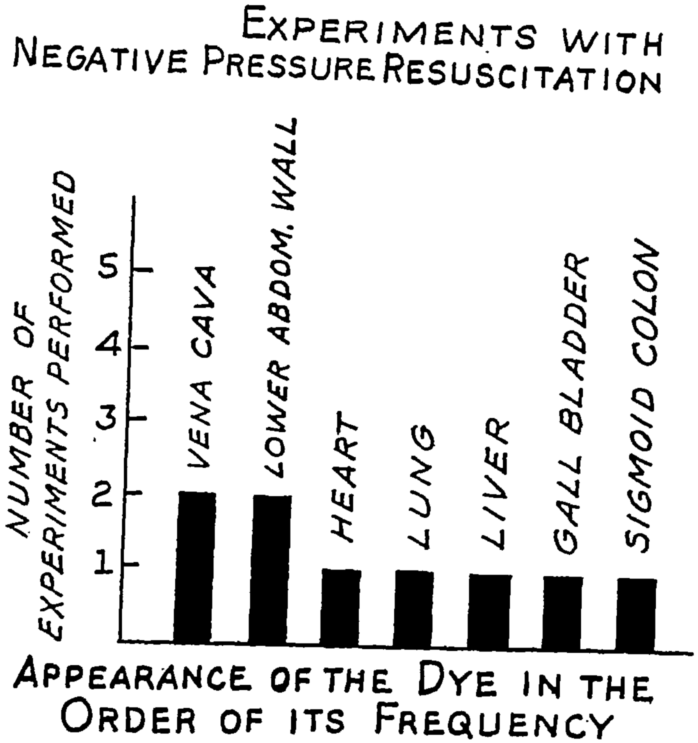
Before autopsy the fluorescein was seen most frequently and earliest within the oral cavity (mucous membranes).

The widest and quickest distribution of the dye occurred in the experiments which were done with alternating positive and negative pressure resuscitation, as can be seen from Charts 1, 2, and 3. Further study of the charts show no appreciable difference between the efficiency of the resuscitation with negative pressure only and positive pressure only, although there were only two experiments in which negative pressure was used.

mucosa (1), diaphragm (1), bladder (1), gall bladder (1), dura mater (1), brain vessels (1), between the toes and under the toenail (1).

Complete autopsies were not made in all animals and that fact accounts for the appearance of the dye reported in the oral cavity and not reported in the other organs. The brain was autopsied in only one animal.

Two experiments were done with negative pressure only. In both occasions the dye was injected into the femoral vein and autopsies performed in forty-five and forty-four minutes, respectively, after the beginning of resuscitation. The dye was found to be limited mostly to the venous side of the vascular system. At autopsies the dye was found in the following sites in order of its frequency (see Chart 2): vena cava (2), lower abdominal wall (2), heart (1), lungs (1), liver (1), gall bladder (1), sigmoid colon (1).



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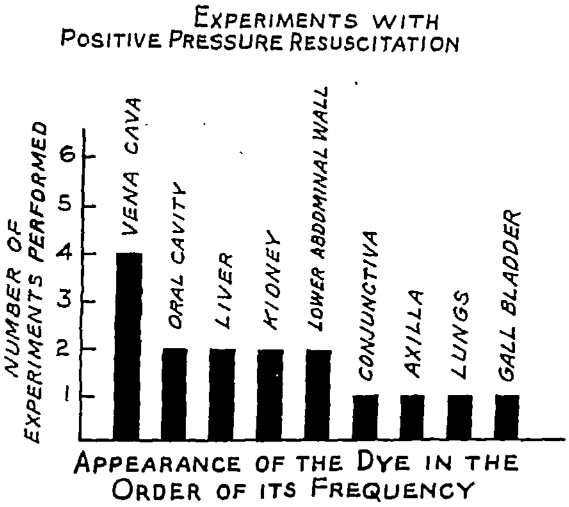

Chart 3.—Appearance of the dye at various sites when positive pressure only was used.

The brain was examined in only one experiment, and this autopsy showed that the brain vessels as well as the brain substance contained large amounts of fluorescein.

To prevent the effect of gravity on the diffusion of the dye, the animals were placed on an inclined plane with the head elevated approximately 10 degrees.

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The control experiments indicate the occurrence of some local and downward diffusion of the dye. They also show the importance of heparinization of the animal, since intravascular clotting takes place soon after death. Once clotting has occurred, no further movement of the blood column is possible. As long as the blood remains liquid it can be circulated, and this means an increase in the possible resuscitation time.

CONCLUSIONS

Inflation and deflation of the lungs in a previously heparinized animal immediately after death produces a movement of the blood column within the vascular system. This can be observed by the intravascular injection of a tracer substance, fluorescein, which gives a typical luminous appearance when examined under an ultraviolet light. The movement of the blood is in a normal direction, and as such can be circulated over the entire body. This movement of the blood takes place without the benefit of any cardiac activity.

The widest and quickest distribution of the dye occurred when alternating positive and negative pressure resuscitation was used.

The importance of heparin in resuscitation is demonstrated. Heparin keeps the blood in a fluid state and as such allows it to be circulated. Once clotting has occurred, no further circulatory movement is possible and all resuscitative attempts will fail.

Resuscitation with alternating positive and negative pressures is superior to resuscitation with positive pressure only or negative pressure only.

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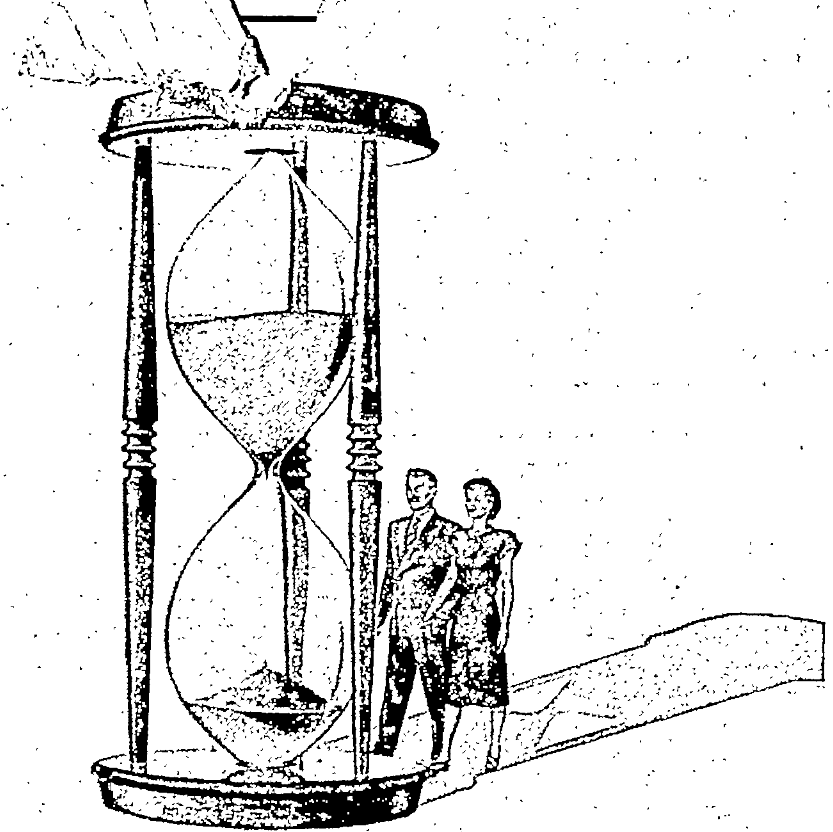

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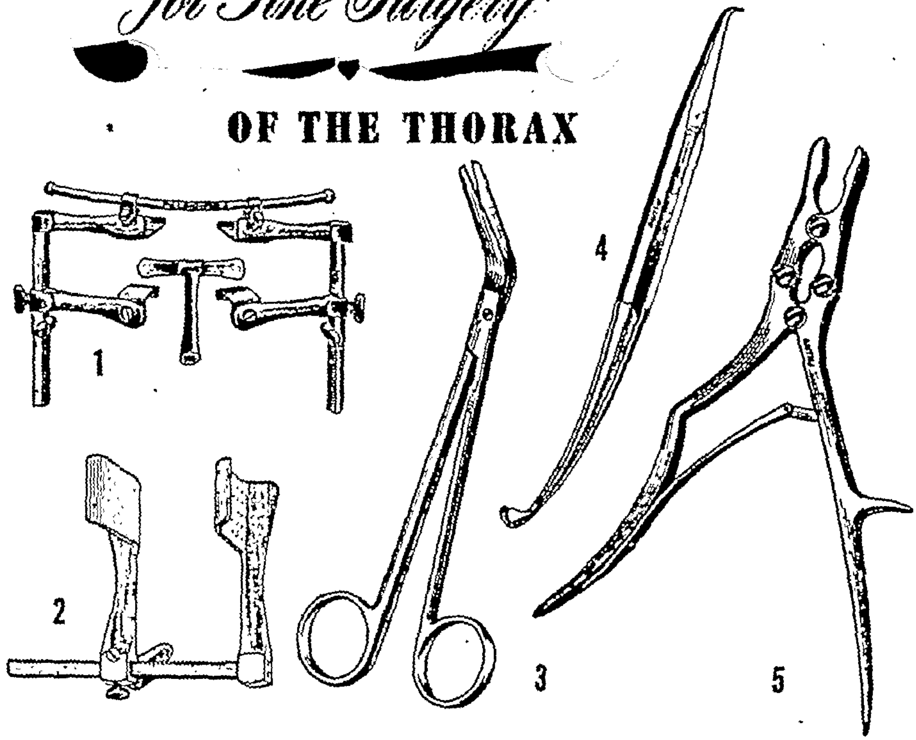

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Original Communications

COMPLICATIONS OF THE SURGERY OF PATENT DUCTUS ARTERIOSUS

JOHN C. JONES, M.D.
LOS ANGELES, CALIF.

THIS report concerns the complications of the surgery of patent ductus arteriosus in a series of sixty-one successive patients. Fifty-three operations were ligations in continuity, and eight were divisions employing the modification of the technique of Gross.[1] All of these patients had a presumably clinically uncomplicated ductus arteriosus, and none of them were believed to have had subacute bacterial endarteritis before operation. The diagnosis of patent ductus arteriosus was clear-cut and was confirmed upon operation in every case.

The youngest patient was just under 3 years and the oldest was 35 years of age. Thirty-three (55 per cent) of the patients were younger than 10 years, twenty-three (37 per cent) were between the ages of 10 and 20 years, and five (8 per cent) were older than 20 years (Table I). The eight patients in whom a division of the ductus was carried out were between the ages of 3 and 6½ years.

TABLE I. AGE INCIDENCE

Age	Under 5	5-9	10-14	15-19	20-29	30-35
No. cases	17 (28%)	16 (26.5%)	12 (19.5%)	11 (18%)	4 (6.5%)	1 (1.5%)

Virtually all of the patients developed some pleural effusion postoperatively, but in only twenty-two (36 per cent) was aspiration deemed necessary, three of whom were aspirated twice. In two of the twenty-two, only air (250 c.c. and 150 c.c.) was obtained. In the remaining twenty, serosanguineous fluid in varying amounts of between 50 and 450 c.c. was obtained by aspiration. Penicillin intrapleurally or intramuscularly, and the sulfonamides intrapleurally or orally were not used, either pre- or postoperatively, except in patients developing a

---

Read at the Twenty-sixth Annual Meeting of the American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

complication indicating their specific need, and in none of this series were these drugs employed as prophylactic treatment.

Postoperative pulmonary atelectasis occurred in five patients (8 per cent), all children, each time in the left lower lobe. Their lungs had been inflated at intervals routinely during the surgery and all atelectatic areas cleared before closure of the chest, the pleural cavities had been aspirated after closure, and inhalations of carbon dioxide had been administered for several days following surgery. All five patients recovered following frequent postural treatment and more vigorous and frequent inhalations of carbon dioxide, and in three cases small doses of sulfonamides were given orally. Penicillin was not available at the time.

Left recurrent laryngeal nerve paralysis occurred three times in this series. In two patients it has persisted, and in one total recovery was noted within a few weeks after operation. In spite of having identified the nerve in every instance at the beginning of the dissection of the ductus and having been careful not to injure it during the operation, this complication occurred. All three were in cases in which the patent ductus arteriosus was ligated and it is very likely that the pressure of the tapes and their knots and the scar tissue about them subsequent to the edema immediately following surgery accounted for the permanent paralysis, for the nerve was intact after the ligation was completed. In the two patients (both adult females) with persistent paralysis, the quality of the voice improved considerably over a period of several months after operation, but the appearance of the vocal cords had not changed.

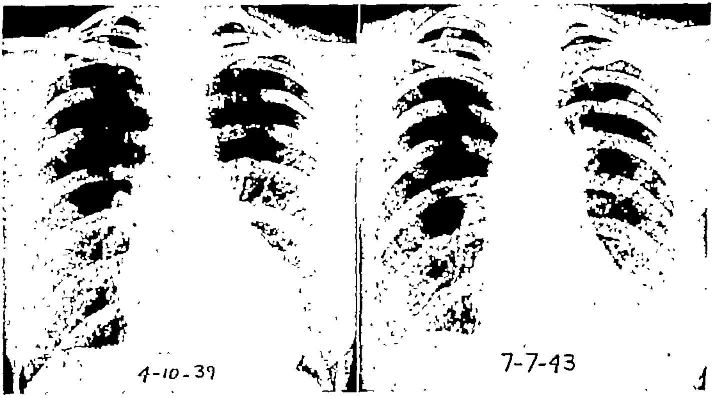
Two of the patients developed an empyema postoperatively. The first was a 16-year-old girl who presumably did not have a pneumonia, but developed a *Staphylococcus aureus* infection in the pleural space after the cultures of the first aspirated fluid were negative. She had a rib resection, and, while the lung was reluctant to re-expand, she finally made a complete recovery from the empyema. The other recently operated patient, a boy of 3 years, developed a pneumonia in the left lung following division of the ductus arteriosus. The lung consolidation and high fever came on thirty-six hours after operation, and he developed an empyema in spite of prompt chemotherapy (sulfadiazine and penicillin). Aspiration and instillation of penicillin into the pleural cavity was employed, but the fibrin clots finally demanded rib resection. He is making a good recovery but the tube is still in place. There was but the one pneumonia in this series.

There were no wound infections. The high incidence of keloid in the supramammary scars has forced us to abandon this approach through the second intercostal space anteriorly. In children as well as adults, if the anterior approach is preferred, the incision should be an inframammary one, for certainly in adult males and children the tension of the pectoralis muscles is the great factor in keloid formation rather than the weight of the breast. In the last twenty cases we have employed a posterolateral approach (resection of fourth or fifth ribs) and we believe it affords a far better exposure for any operation in the area of the ductus arteriosus. All our divisions have been carried out through this approach, and the incidence of keloid in the scars has been greatly diminished.

A serious distressing complication following ligation of the ductus arteriosus in our experience has manifested itself by sudden copious hemoptysis. Two case histories are briefly outlined with their follow-up.

CASE REPORTS

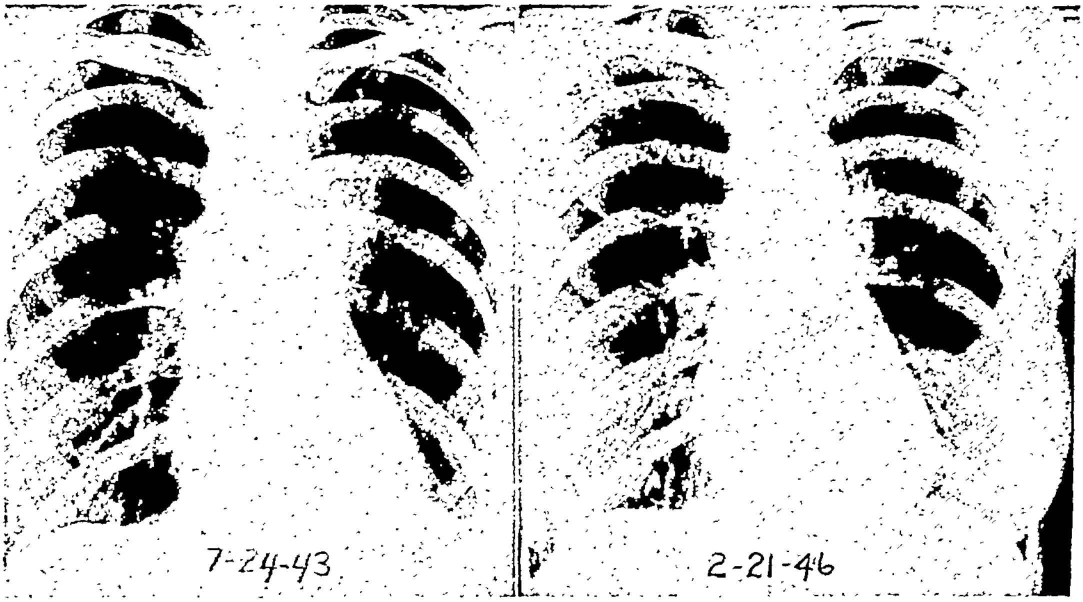
A. J., 35-year-old housewife, had an uneventful convalescence following ligation of the ductus arteriosus Feb. 22, 1943, and was discharged from the hospital thirteen days later. On July 3, 1943, after a strenuous day of housecleaning, she coughed up an ounce of bright red blood. The next night she awakened with rattling in her throat and had a similar experience with hemoptysis. The following two days she had an aching across and between the shoulders and raised more dark bloody mucus plugs before being admitted to the hospital July 7. She said she felt well otherwise. The temperature, pulse, and respirations were normal and the blood pressure was 120/90. Examination revealed a heart that was definitely smaller than before operation and now within the limits of normal and only a slight pulmonic systolic murmur could be heard. The blood counts were well within the normal range and electrocardiogram was not abnormal. The roentgenogram (Fig. 1, *A* and *B*) revealed a slight haziness in the left upper lung field and a slightly increased bulging of the left upper mediastinal border in the region of the pulmonary conus, with a normal contour of the heart. Roentgenogram twenty days after the onset of hemoptyses disclosed an increase in the mediastinal bulge and diffuse haziness in the medial aspect of the left upper lobe (Fig. 2, *A*). She remained in the hospital for six weeks with occasional small hemoptyses, almost daily streaking of mucus and some occasional aching pain between the spine and the left scapula above the fourth dorsal vertebra, but was discharged home in good condition to remain in bed. On Nov. 19, 1943 she had a sudden hemoptysis of 350 c.c. and was readmitted to the hospital. She was not in shock, the pulse was 100, blood pressure 130/80, and hemoglobin estimation was 80 per cent. Physical examination revealed râles over the left lung field and a persistent pulmonic systolic murmur, but no continuous or diastolic murmur. Roentgenogram and electrocardiogram remained the same. She had another hemoptysis of 200 c.c. of bright blood three days later, and was discharged eleven days after admission with no com-



A. *B.*

Fig. 1.—*A*, roentgenogram of A. J. before operation showing prominence of the pulmonary conus and enlargement of heart. *B*, roentgenogram four days after onset of hemoptyses. Bulging in the area of the pulmonary conus and haziness of the mediastinal aspect of the left upper lobe is noted.

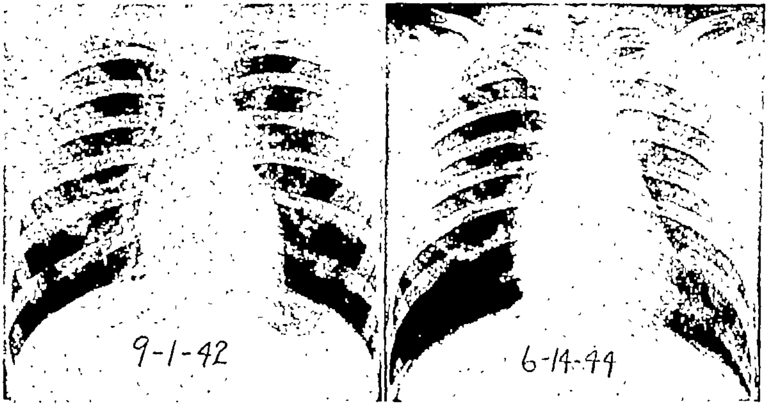
plaints other than a little irritative cough and occasional streaking. A week later she had a massive sudden hemoptysis of 500 to 750 c.c. at home, and a transfusion was administered the following day because of a hemoglobin estimation of 63 per cent and 3,840,000 red blood cell count. On Jan. 8, 1944, another 500 c.c. hemoptysis occurred, then only streaking occasionally until February, 1945, when she had a 200 c.c. hemorrhage and none since then. She was last seen on May 17, 1946, and appeared well (Fig. 2, *B*). She volunteered that she had occasional streaking of her mucus in the morning, had led a normal active life with no limitation for over a year with no frank hemoptyses, and weighed 136 lbs. Pulse was 85, blood pressure 118/80. She had a low-grade systolic pulmonic murmur while sitting which disappeared in the supine. The lung fields were clear to auscultation. Fluoroscopy revealed the heart to be normal in size and contour, the lung fields were clear throughout; there was no conus fullness, and the previous bulging of the left upper mediastinum had disappeared. Electrocardiogram and blood counts were within the normal range.



A. *B.*

Fig. 2.—*A*, roentgenogram of A. J. twenty-one days after initial hemoptysis showing increased mediastinal bulge and upper lobe opacity. *B*, roentgenogram one year after the last hemoptysis. Patient felt well.

J. S., female, 11½ years old, had her patent ductus arteriosus ligated in continuity with two silk umbilical tapes on Sept. 14, 1942 (Fig. 3, *A*). Convalescence was uneventful, there remained no cardiac murmurs, and she was discharged in excellent condition thirteen days later. She remained well without symptoms and without abnormal physical findings on examination until June 13, 1944 (twenty-one months after operation), when she coughed up a mouthful of bright red blood. She did not feel ill and had no other symptoms, but was admitted to the hospital for study. Temperature remained normal. The pulse was 100, and respirations 20. Blood pressure 140/100 on admission receded to 96/68 the following day. Moist râles were heard in the left lung, but cardiac findings and electrocardiogram were normal. X-ray of the chest (Fig. 3, *B*) on admission showed slight haziness of the left upper lobe which had cleared in forty-eight hours. Three hours after admission, while lying quietly in bed, she had a sudden hemoptysis of 4 oz. of bright blood. She was discharged after three days' observation. The following day, June 18, 1944, she had another 4 oz. hemoptysis and x-ray immediately afterward again showed only a slight haziness of the left upper lobe which cleared in forty-eight hours, and the physical findings were as before. Bronchoscopy June 22 was negative, as was the examination of the heart and lungs. She had six recurrences of the hemoptyses (Fig. 4, *A*) the last of which amounted to 500 c.c., and she was admitted to the hospital Nov. 1, 1944, with temperature 100.2° F. (rectal), pulse 84, respirations 20, and blood pressure 94/56. She had a soft systolic murmur at the apex, but no other murmur. The left upper lung was slightly hazy by roentgenogram. The hemoglobin was 64 per cent, red blood cell count 3,470,000, white blood cell count 11,450. On November 2, the thorax was explored through a posterolateral approach (resection of the fifth rib) under endotracheal ether-oxygen anesthesia. The lung was adherent to the anterior chest wall at the site of approach for the ligation of the ductus arteriosus. A knuckle of lung, 2 cm. in diameter (mediastinal aspect of the left upper lobe) was densely adherent to the aorta at the site of ligation of the patent ductus arteriosus, but there was no thrill. It was as though the lung had been drawn into the hollow of the arch of the aorta, adjacent to which was an area of indurated firm lung the size of a walnut. While dissecting the lung from the aorta traversing recent and old organized blood clot, granulomatous appearing tissue exuded and two silk (umbilical tape) knots popped out (Fig. 4*B*). When completely freed from the aorta there was seen a 2 cm. cavity in the lung which connected with an open bronchus. Suddenly a forceful, 4 mm. stream of blood came from the aorta just beyond the site of the ductus ligation and from the granulomatous appearing area of the aorta. This was immediately controlled by digital pressure. It was obvious that the two silk knots had eroded both the aorta and the lung and that a fistulous tract between the aorta and bronchus had been established. Five fine (00000) silk sutures in the wall of the aorta closed the opening and this was reinforced by suturing over it a layer of mediastinal pleura. The bronchial opening and the cavity in the lung were obliterated with fine catgut sutures on atraumatic needles. The lung was re-expanded, 2 Gm. of sulfanilamide crystals and 25,000 units of penicillin in 50 c.c. of saline were left in the pleural cavity, and the chest wall was closed without drainage. The blood pressure before operation was 90/60, at the close it was 90/70. During the latter part of the operation, 250 c.c. of blood was given. She had an uneventful convalescence and was discharged Nov. 7, 1944, fifteen days after operation (Fig. 4*C*). She has been well ever since and has no abnormal physical findings referable to heart or lungs (Fig. 4*D*).



A. *B.*

Fig. 3.—*A*, Roentgenogram of J. S. before operation. *B*, Roentgenogram a few hours after initial hemoptysis, showing bulging at the conus arteriosus and haziness of the left upper lobe.

We believe that both the above patients had identical lesions, an aortobronchial fistula, in spite of the fact that the first was not proved by operation.

In both there was no recurrence of the continuous murmur and no evidence of a fistulous tract from the pulmonary artery to the aorta, and the patency of the ductus has surely been obliterated. Both patients had similar clinical histories and roentgen findings. The magnitude of the hemoptyses in the first patient makes it unlikely that the silk knots eroded only the lung and that the bleeding could be from the lung itself. In spite of the fact that the patient has not had frank hemorrhage for fifteen months and the lesion appears to be healing, the fact remains that occasional streaking in the mornings occurs and there may be recurrences requiring surgical intervention.

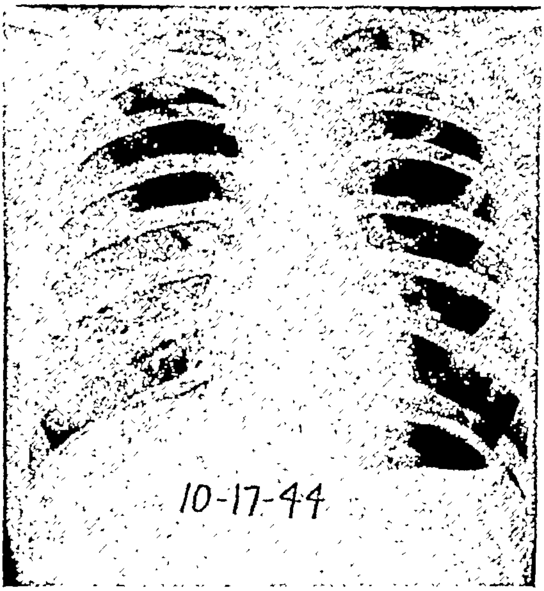


Fig. 4A.—Roentgenogram of J. S. four months after onset of hemoptyses.

The mediastinal pleura over the site of the ligation of the ductus with two silk umbilical tapes tied side by side was not closed or sutured in either patient, and it may be that if it had been closed, this complication could have been avoided. The mediastinal pleura has been closed, burying the knots in all, since this experience. This procedure would protect the mediastinal aspect of the upper lobe but would not prevent erosion of the aorta or possible aneurysm. At any rate, this complication is a definite argument in favor of the division of the ductus arteriosus.

There were two deaths in this series. The first patient (J. D.) operated May 6, 1939, was discharged from the hospital fourteen days later following an uneventful recovery. She resumed normal activity and thirty days later developed a high fever and died of *Staph. aureus* endarteritis forty-eight days following ligation. At autopsy, a re-established fistula between the pulmonary artery and the aorta, around the ductus ligatures, and an aneurysm at the ductus were found. The ligatures had cut through the wall of the ductus arteriosus and a superimposed blood stream infection resulted in vegetations on both the pulmonary artery and aortic walls. The details of this case were presented at the Los Angeles meeting.[2]

The other death was an operative mortality. W. W., male, 6 years old, was diagnosed as having patent ductus arteriosus on his admission to the Children's Hospital in 1942 for the treatment of pneumonia. Roentgenograms revealed bilateral pneumonia and a large, round heart consistent with a congenital lesion, and a fullness of the conus arteriosus. He made a rapid recovery and was observed for over three years. He was thin and underdeveloped and had not been able to carry on with others his own age. On admission to the hospital April 20, 1946, the pulse was 108, and blood pressure 92/38. Examination revealed a symmetrical chest that bulged forward, and a very active precordium. The lungs were clear. The point of maximum impulse was in the fifth interspace at the anterior axillary line, and there was a great continuous thrill over the pulmonic area. A loud, harsh, continuous machinery murmur over the pulmonic area was transmitted over the entire precordium and could be heard throughout the chest. His roentgenogram revealed a moderate cardiac enlargement with clear lung fields. Electrocardiogram before admission was not remarkable, and his blood counts were normal. On April 22, at operation, it was noted that the cardiac action was considerably more vigorous than was usual in these cases and the ductus arteriosus stood out more prominently and seemed stiffer, firmer, and thicker than the average ductus we had seen. Dissection around the ductus arteriosus was completed easily and while not being disturbed, the posterior pulmonary artery end of the ductus gave way with a resultant large hemorrhage. Two tapes passed around the ductus and tied failed to control the bleeding, but after forceps were applied, the bleeding stopped, the tapes were removed, and the ductus was divided. The aortic aspect of the ductus was sutured with fine silk and that forceps removed. One small leak in the aorta was closed with an interrupted suture. The pulmonary side of the ductus was then sutured distal to the forceps. Blood transfusion had been instituted at the onset of the hemorrhage. When the forceps was removed from the pulmonary side of the ductus, there was another, larger gush of blood proximal to the suture line which was fairly well controlled by digital compression while two interrupted mattress sutures were taken in the leaking pulmonary artery. This completed hemostasis, but at this point cardiac arrest intervened, and in spite of the usual restoration measures, we were unable to stimulate cardiac action. He had lost considerable blood at a rapid rate on two occasions, and we were unable to replenish the loss quickly enough to resuscitate the heart. The more pertinent findings at necropsy are outlined. The heart weighed 212 Gm. (normal, 94 Gm.) and was considerably enlarged. There were areas of pale, white, thickened epicardium over

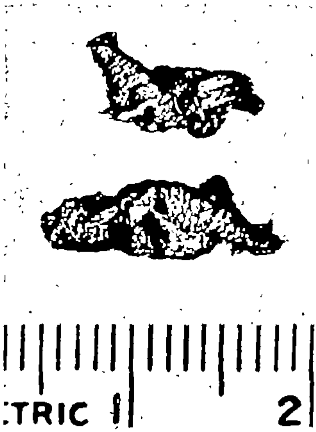
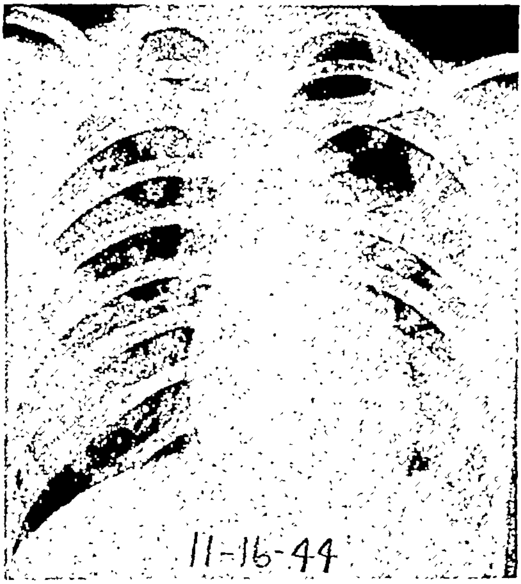


Fig. 4B.—Two silk umbilical ties and knots encased in fibrous tissue removed at operation. Patient J. S.

the apex and right ventricle. Numerous small verrucose nodules were noted over the left auricular appendage, having the appearance of vegetations. The myocardium was increased in thickness, the papillary muscles hypertrophied, and the chordae tendinae were thicker and less transparent than usual. The endocardium was smooth and glistening, except for that over the mitral valve and immediately above it. Here the endocardium was thickened and not transparent, and was white. The mitral valve was increased in thickness, and had a nodular appearance and feel to it. The foramen ovale was 2 mm. in patency. The ductus arteriosus was absent at its two points of origin in the pulmonic and aortic aspects. It had apparently recently been closed and numerous fine sutures were in place. The inflammatory lesions of the heart, exclusive of the congenital lesions, were reported as consistent grossly and microscopically with rheumatic heart disease, although no typical Aschoff's bodies were found. Blood cultures were negative in ten days. The pathologist's diagnosis was cardiac hypertrophy and dilatation, chronic mitral endocarditis, pericarditis, and focal myocarditis.



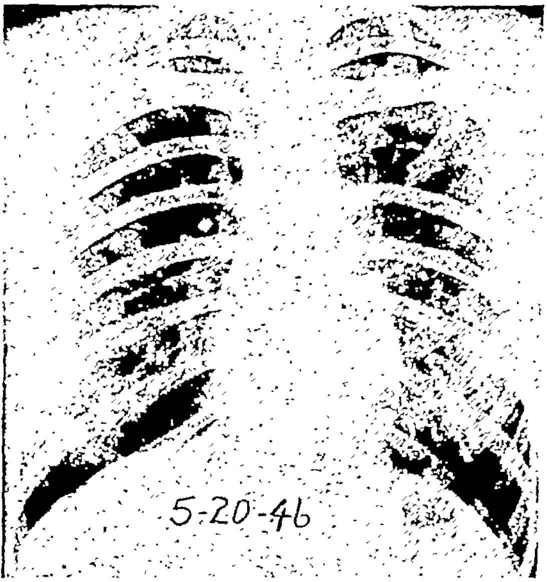


Fig. 4C.

Fig. 4D.

Fig. 4C.—Roentgenogram of J. S. on discharge, two weeks after exploration and repair of aortobronchial fistula.

Fig. 4D.—Roentgenogram of J. S. eighteen months after discharge from hospital.

The ligation or division of the ductus arteriosus, had it been successful, might possibly have decreased the myocardial load and lengthened this patient's life, but we were ignorant of his having rheumatic heart disease at the time of his operation; we would not recommend surgery for patent ductus arteriosus complicated by rheumatic fever.

While this, the only operative death in the series, was a division of the ductus arteriosus rather than ligation, the death cannot be attributed to the division per se, for the end result would probably have been the same regardless of the method employed. The ligation with tape utterly failed to arrest the hemorrhage, but it was controlled by the division of the ductus arteriosus, and in retrospect, in this instance at least, it would have been judicious to have proceeded with division at once.

Probably the most embarrassing complication of the ligation of the patent ductus arteriosus from the standpoint of the surgeon is the persistence or recurrence of the continuous murmur. In fifty-three ligations, there were five patients (9.4 per cent) whose continuous murmur was noted within the first few days after operation (as soon as the dressings were removed and an examination was permitted). The persistence of the murmur can be explained only by a failure to tie the tapes tightly enough to occlude the lumen of the ductus completely. Five others (9.4 per cent) were discharged from the hospital either with no murmur or a systolic pulmonic murmur, only to turn up later with a recurrence of the continuous murmur and usually some palpable thrill. In this group, the murmur was detected five or six weeks after ligation when the patient returned for examination, and presumably is explained by re-establishment of the fistula after the tapes had cut through the wall of the ductus arteriosus. We have not operated a second time on any of these ten cases. In some the murmur has remained unchanged, in others it has increased or diminished in its intensity; in seven of the patients, the murmur is *now not as* loud as before surgery, in two it is about the same as before ligation, and the remaining patient (J. D.) is dead.[2] In the seven cases surviving the division of the ductus arteriosus, there have been no recurrences of the murmurs nor any complications attributable to this method of treatment.

There are fifty-nine (96.5 per cent) patients alive, nine of whom have a persistent continuous murmur after the ligation of the ductus arteriosus. All of these patients are well with one exception, an adult female with an early recurrence of the murmur, a persistent vocal cord paralysis, diabetes, and a low reserve.

SUMMARY

1. Sixty-one cases of patent ductus arteriosus have been operated on; fifty-three (87 per cent) were ligated in continuity, and eight (13 per cent) were divided and sutured.

2. There were two deaths, one a late death due to *Staph. aureus* endocarditis, and one operative death (exsanguination and cardiac arrest).

3. Two patients developed an aortobronchial fistula with large hemoptyses, one of which was repaired surgically; the other appears to be healing spontaneously.

4. Five of the ligations had an early recurrence of the murmur due to incomplete occlusion of the vessel by the tapes, and five others had a late establishment of the fistula due to the tapes partially cutting through the wall of the ductus.

5. Five had left lower lobe postoperative atelectasis.

6. There were two empyemas, one of which followed a pneumonia.

7. Two patients have a permanent, left, recurrent, laryngeal nerve paralysis.

8. Fifty-nine patients are alive, and, in spite of nine patients having a persistent continuous murmur, only one is not well. She is a diabetic with a reestablished fistula and a low reserve.

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COMPLETE DIVISION FOR THE PATENT DUCTUS ARTERIOSUS

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DURING the last eight years, studies from this and other clinics have amply demonstrated that surgical therapy is an important method for the treatment of individuals with a patent ductus arteriosus. In the early stages of these surgical endeavors, closure of the ductus was always attempted by some form of ligation. It was fully realized that ligation of a vessel "in continuity" had certain limitations, because this procedure might not give complete closure of the shunt, or else there might be partial recurrence of the fistula if the ligatures cut through the vessel wall. While it was obvious that complete division of the ductus would be desirable, limited operative experience at that time made me feel that this was too hazardous an undertaking. Therefore, ligation alone was done, and in the great majority of cases this gave a good result and could be accomplished with a relatively low risk.

In the first part of the work, forty-three individuals of varying ages and with manifold complications of a persistent ductus were operated upon by some form of ligation. In most of these, two ligatures of No. 8 braided silk were used. In a few instances, narrow umbilical linen tape was employed. In twenty-nine cases, double ligatures were placed and then Cellophane was wrapped over these, the author believing that subsequent fibrosis excited by this material would cause some regional scarring and close off any small shunt which might have been left at the end of operation. The addition of a cellophane wrapping was an improvement, but it could not be relied upon to give a permanent closure of the shunt in all cases. A careful follow-up study on these forty-three patients (forty of whom survived surgical treatment) shows that in general, about 80 per cent obtained complete closure of the ductus and that the vessel remained closed. In about 10 per cent of the cases there was apparent complete closure of the ductus at the end of the operative procedure, and for a week or ten days thereafter, but subsequently the ligatures partially cut through the ductus and some of the fistula was re-established. In the remaining 10 per cent of the cases, the ligatures were not tied quite tightly enough, so that a small shunt was left at the close of the operation. In these last two groups the results were somewhat disappointing, but this does not vitiate the fact that most of these individuals were greatly improved because the work of the heart was tremendously reduced, and in some cases a *Streptococcus viridans* infection was permanently cured. While this general therapeutic picture might be classed as quite satisfactory, an attempt has now been made to develop a technique for complete division of the ductus in

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all cases. This goal appeared difficult to attain, but the search was stimulated by the conviction that division of the ductus would be the only operation which could give assurance of a complete and a permanent closure of the shunt.

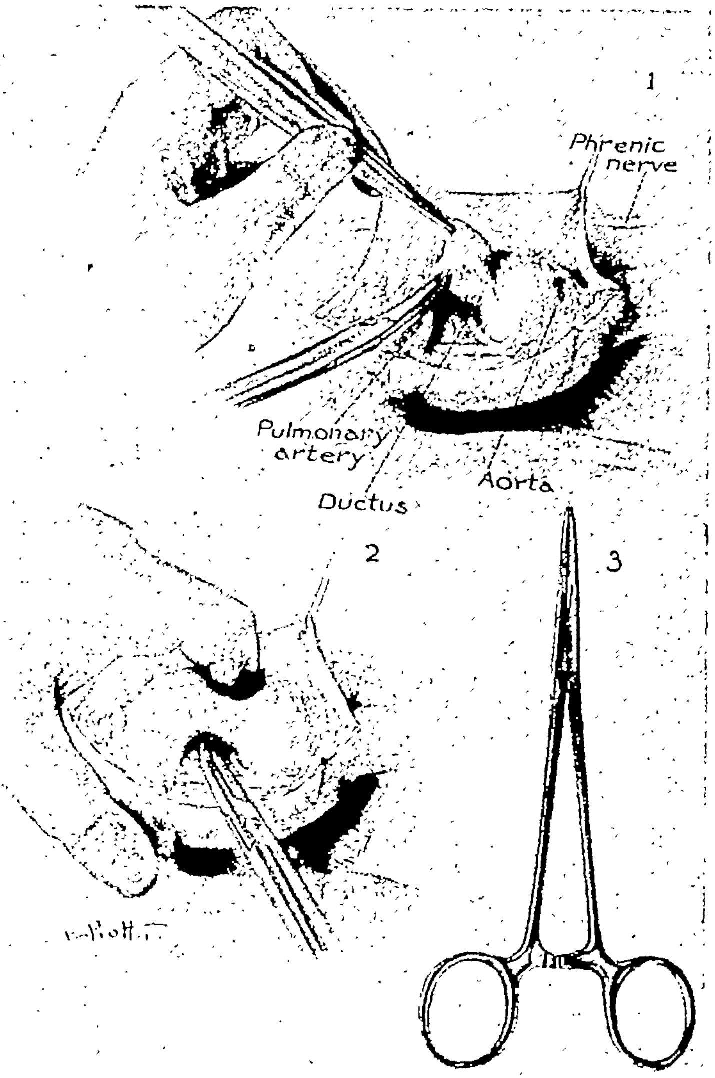
As far as I am aware, Touroff[3] was the first (Jan., 1940) to divide completely a patent ductus arteriosus. In 1941, I divided the ductus in a child of 10 years, and in 1944 reported the technique[4] which had been successfully used in fourteen cases. To date, I have divided the ductus in 90 patients, and would like to indicate briefly the technique which is currently employed. This is essentially the same type of operation, with but minor changes, as that which was described in the publication of 1944.

OPERATIVE TECHNIQUE

A route for exposure of the superior mediastinum and great vessels has been rather fully considered in other communications.[2, 3] Only a few points will be stressed here regarding this approach. The patient is placed on the table with a sandbag underneath the left side of the chest and left shoulder. The left arm is extended upward along the head. A long, curved incision is made beneath the left breast extending well around to the posterior axillary line. The entire breast is dissected free and raised from the pectoral fascia so that the breast can be turned upward, a point of extreme importance in gaining an adequate exposure in adult females. The pectoral muscles are divided just above their attachment to the thoracic cage. The anterior border of the latissimus dorsi muscle must be severed for an inch or two. The anterior serratus muscles are cut as far back as the long thoracic nerve. The left pleural cavity is entered through the third intercostal space, the intercostal muscles being divided from the sternal border around almost to the angle of the ribs. The third and second costal cartilages are cut so that the ribs can be pushed upward and held in that position with self-retaining retractors. It is extremely important to obtain a maximum opening from these various steps, in order to have sufficient room when working inside the chest.

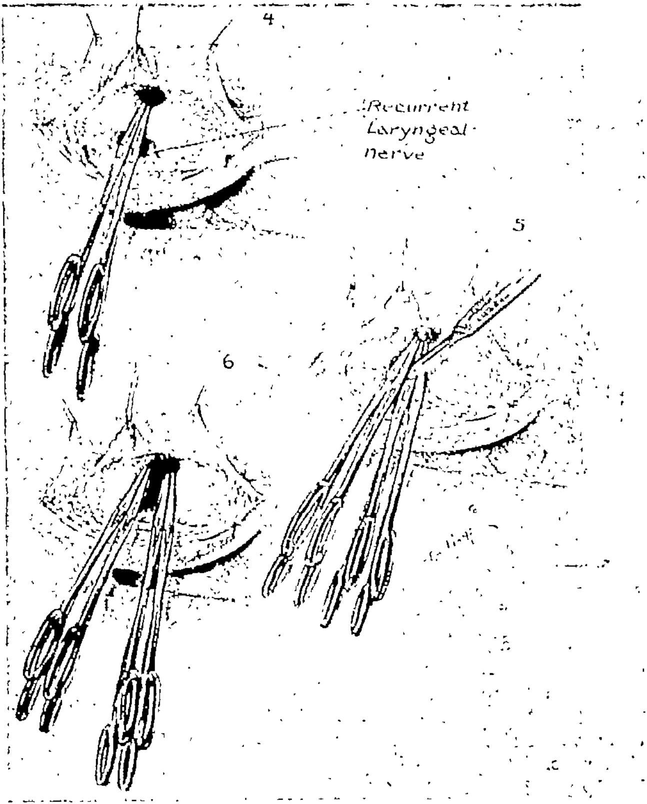
The superior mediastinum is entered by a long cephalocaudal incision of the parietal pleura, this cut being made about one-half inch behind the phrenic nerve. The highest intercostal vein which courses over the mediastinal structures to reach the left innominate vein must usually be divided. The vagus and left recurrent laryngeal nerves are carefully searched for and spared from injury. Lymph nodes which obscure the field must be dissected away before exposing the underlying structures. Fat and areolar tissue must be cleared away from the terminal portions of the aortic arch and the near-by pulmonary artery so that the intervening ductus can be clearly visualized. In most subjects, a small lappet of pericardium extends upward and partially overlays the left lateral surface of the ductus. This must be completely removed from the ductus (so that it will not be subsequently included in the clamps which are placed upon the ductus). By sharp and blunt dissection (Fig. 1) this lappet can be pushed away. It is well to complete this step without perforating the pericardial sac; this precaution prevents pericardial fluid from running into the field.

The posteromedial wall of the ductus is now circumvented by blunt dissection, which is most easily done with a right-angle clamp (Fig. 2), guiding the instrument by feeling the point with the tip of the left index finger. In this way, the right-angle clamp is carefully pushed between the wall of the ductus and the left main bronchus. After a hole has been made behind the ductus in this manner, the opening can be carefully enlarged by sharp dissection under direct visualization. Behind the back wall of the ductus there is usually a web of fibrous tissue which fans downward to the pulmonary artery, medially to the bronchus, and superiorly to the medial surface of the aorta. This substance



Figs. 1, 2, 3.—*1*, Lappet of pericardium being turned caudally by dissecting it off of the ductus arteriosus. *2*, Back (medial surface) of ductus being freed by blunt dissection. *3*, Type of instrument to be subsequently used for clamping the ductus. The jaws have been ground so that they are thinner than stock instruments.

must be cut away adequately to give as much room as possible on the medial surface of the ductus. This dissection brings one dangerously close to the pulmonary artery, the left main bronchus, and the undersurface of the aortic arch, but in no case of the present series was any one of these structures injured.



Figs. 4, 5, 6.—4, Ductus completely uncovered. Two clamps applied. 5, Four clamps in place. Ductus being cut between the two middle clamps. 6, Ductus completely severed, leaving two clamps on either end.

Touroff[6] has emphasized that sometimes it is possible to get into a tissue plane which allows the operator to dissect more freely around the ductus. All of the ductus dissection and liberation must proceed with great caution and must be completed with thoroughness, because this single step is most important in facilitating subsequent stages in complete division of the vessel.

Fig. 3 represents the type of instrument which has been used for clamping of the ductus. This is a standard Crile clamp, 5½ inches in length, the jaws of which have been ground off on both sides so that the surfaces are nearly parallel and are thinner than stock instruments. In the first eighteen cases in which complete division of the ductus was performed, each of these clamps was fitted with a rubber band on the handle so that the instrument could be placed on the ductus and held in that position by the tension imparted by the rubber band. This precaution was taken so that there would be no undue crushing injury to the ductal wall. In all of the subsequent cases, this elastic band has been discarded and each clamp has been placed on the ductus so that only its first ratchet is engaged. In no instance has this caused any important trauma to the ductus wall, nor has it led to subsequent sloughing or bleeding in any patient.

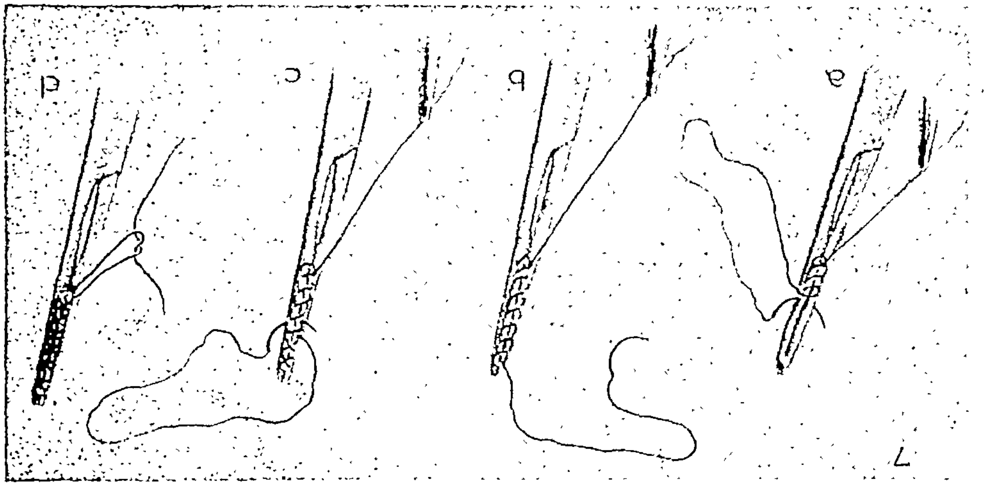
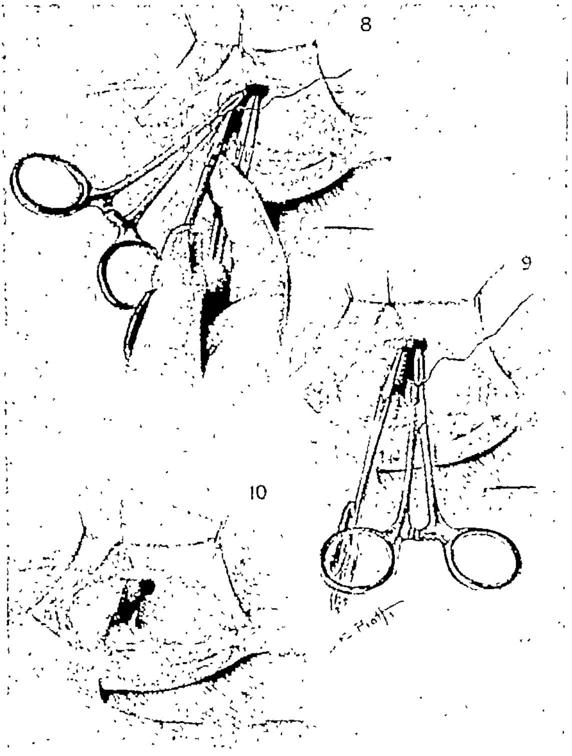


Fig. 7.—Details of closure of pulmonary end of ductus. One clamp has been removed, leaving a cuff which is sewed over and over with fine silk suture.

After the ductus has been completely freed of areolar tissue and has been cleared of any investing membrane, two clamps are applied as shown in Fig. 4. In most patients this appears to take up all of the room between the aorta and the pulmonary artery. However, it is possible (Fig. 5) to squeeze on a third clamp which rides up onto the pulmonary artery and a fourth clamp which rides up onto the aorta. With these four clamps in place, the ductus is cut in half by a scalpel which is run between the two middle clamps. This, then, leaves (Fig. 6) the divided ductus with two clamps on its pulmonary end and two clamps on its aortic end. It is at this stage that the above-described dissection will pay dividends, because the previous freeing of structures medial and posterior to the ductus will now allow the pulmonary artery and aorta to separate from one another and give the operator room to work on the individual ends of the severed ductus.

Fig. 7 shows details of closing the pulmonary end of the ductus. The presenting clamp (the one nearest the operator) has been removed and thus provides a small cuff of ductus wall which is 2 or 3 mm. in length. This can be whipped over and over with a continuous 5-0 Deknatel silk suture carried on a small atraumatic needle. When the end of the ductus has been closed in this way, the remaining end of thread is brought back and tied to its original end.



Figs. 8, 9, 10.—*8*, Cuff on pulmonary end of ductus being closed in the manner shown in Fig. 7. *9*, Pulmonary end of ductus closed. One clamp is then removed from aortic end and the cuff of tissue will be sewed over and over, the stitch passing through the full thickness of ductus wall. *10*, Division of ductus completed. Individual closure of aortic and pulmonary ends of ductus.

Fig. 8 demonstrates the step of closing the pulmonary end of the ductus after the presenting clamp has been removed and a cuff has been made. In Fig. 9 a similar treatment is being carried out for the aortic end of the ductus, from which the uppermost clamp has been removed. The cuff thus provided is being sewn over and over with a continuous suture in a manner similar to that which was used for the pulmonary end of the ductus. After the suturing of pulmonary and aortic ends has been completed, it is customary to squeeze a gauze sponge down between the aorta and pulmonary artery, and while this gauze is in place, the remaining pulmonary clamp is removed. The pack is held in place for several minutes to allow blood to coagulate between the interstices of the sutured end of the ductus. The pulmonary end is then inspected, and when it is found to be completely dry, another gauze pack is jammed in between the aorta and pulmonary artery and the remaining aortic clamp is removed. This pack is also held in place for several minutes to allow clotting to occur between the stitches in the aortic end of the ductus. The pack is then removed, and in the vast majority of cases the field is completely dry. In only a few cases has it been necessary to take an extra stitch or two to close some pinpoint opening. (In the first eighteen cases of this series, a second line of sutures was employed to bring together the adventitia of the pulmonary artery to re-enforce the first layer of sutures. A similar adventitial closure was likewise done on the aortic side. Later experience has shown that these second layers are not necessary.) Fig. 9 shows the condition of the aorta and pulmonary artery after all instruments have been removed and the two ends of the ductus have been individually closed. It is probably not essential to do so, but it has been my routine to close the mediastinum by repairing its parietal pleura with interrupted silk stitches.

It is unnecessary to enumerate here the steps for closure of the thoracic wall wound, which steps have been amply treated in a previous communication.[4] It is, however, well to emphasize that careful closure of the chest is of extreme importance in reducing the postoperative discomfort of the patient to a minimum, in producing a cutaneous scar which is not unsightly, and in reducing the postoperative wound complications to a negligible figure.

COMMENTS

In a group of ninety individuals who have been treated by complete division of a patent ductus arteriosus, the ages ranged from 2 to 46 years. In this series there have been two surgical deaths.* One fatality occurred two weeks after operation from a staphylococcus mediastinitis and overwhelming infection. The second occurred in a 26-year-old woman with an advanced stage of cardiac decompensation; division of the ductus had been completed, but the patient expired shortly thereafter with cardiac dilatation and failure. Thus, there have been no deaths which are attributable to ductus division itself. Hence, in the experience of this clinic, complete division of the ductus has not carried any higher operative risk than ligation of this vessel.

Without question, some individuals from this group of ninety patients could have had a perfectly satisfactory result from ligation. Possibly it would have been good judgment to ligate those ducti which were small, soft, and easily compressible, reserving the division technique for the more difficult cases wherein ligation would probably fail to give a complete and permanent closure. However, from a practical point of view, it has been better to use only one technique so that the surgical team could be accustomed to a single, routine procedure, and could standardize this thoroughly. In this way the operator and team have adequate rehearsal, and hence are better equipped to tackle the occasional case in which there is a formidable problem because of the conditions in the ductus or the regional vessels.

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*See addendum.

In these ninety individuals with complete division of the ductus, there were three with superimposed *Str. viridans* infection. While division was undertaken with some trepidation because of the friability of the walls of the pulmonary artery or aorta, in no case did hemorrhage occur at the time of operation or subsequent thereto. (In the total series of 133 patients with ligation or division of a patent ductus arteriosus, there were eleven instances of superimposed, active *Str. viridans* infection. These were all in the prepenicillin era. In no case could the blood stream be sterilized preoperatively with the sulfonamides which were then available. Of these eleven patients, four continued to have bacteremia following operation and subsequently died from continuing infection. The other seven have been cured of their disease.)

There are doubtless some surgeons who will maintain that division of a patent ductus carries an unwarranted risk, and that adequate results have been obtained by ligation alone. I would like to anticipate these objections by pointing out that personal communications from a considerable number of physicians and surgeons indicate that there are postoperative recurrences in a considerable number of patients. This information has not yet appeared in publications. Furthermore, any claims for completely satisfactory results following ligation should clearly indicate what types of cases have been operated upon. I fully agree that in children, where the ductus is soft, yielding, elastic, and easily compressible, a ligation can almost always be relied upon to produce a permanent closure. However, in older individuals, particularly in adults beyond 18 to 20 years of age, the ductus is frequently unyielding, is difficult to compress, and has an exaggerated pulsation transmitted to it by a sclerotic and fixed aorta. Under such circumstances I am convinced that ligation in any form will occasionally lead to partial recurrence of the fistula. It is primarily because of some disappointments in the adult group that I have turned to division of the ductus in all cases.

SUMMARY

The surgical technique herein described for complete division of the patent ductus arteriosus has been employed in ninety cases. Two patients died, one from staphylococcus mediastinitis and the other from cardiac decompensation. Neither fatality was related to the division itself, and in neither instance would simple ligation have avoided a fatal issue. The complete division of the patent ductus has been so completely satisfactory that all forms of ligation of the vessel have now been completely abandoned.

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ADDENDUM

July, 1947. This series has now grown to 180 cases of complete division with a total of four deaths: one from *Staphylococcus mediastinitis,* one from faulty anesthesia, two from cardiac failure. There have been no deaths from hemorrhage, either at operation or subsequent thereto.

DISCUSSION ON "COMPLICATIONS OF THE SURGERY OF PATENT DUCTUS ARTERIOSUS" BY DR. JOHN C. JONES* AND "COMPLETE DIVISION FOR THE PATENT DUCTUS ARTERIOSUS" BY DR. ROBERT E. GROSS

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—When I was here last in 1939, I had an opportunity to see the first of these cases of Dr. Gross in Boston, and they really intrigued me. Upon returning, I talked to our physicians in Stockholm, and they were not enthusiastic over sending such patients for operation at that time, so it took me about two years to persuade them to try it.

At that time there was already one slight recurrence among Dr. Gross's patients. One could hear a continuous murmur, which was very much reduced compared with the murmur that the child had before operation. We discussed the possibility of dividing the ductus because of this recurrence at that time. In my first operation, a rather wide and short ductus, I divided. You have seen the technique, which was very similar to the one Dr. Gross has described to you just now.

The next few patients had rather long and narrow ducti which I did not consider it necessary to divide. I ligated them doubly and had about 4 or 5 mm. of the ductus between the two ligatures, one up on the wall of the pulmonary artery and the other close to the aorta. I hoped that scar tissue would develop.

Since that time we have operated upon seventy-one patients, and have found that a little more than half of them have had ducti which had such a tough wall and were of such length that they could be ligated easily and safely. The other half, on the other hand, had a short and wide ductus, and those we have divided.

In a borderline patient with not very good general condition, it seemed possible to ligate the ductus, and, wishing to get out of that chest as soon as possible, I did a ligation. When the ligature was tied, it cut through, and bleeding from both the pulmonary artery and the aorta occurred at the same time. It was somewhat difficult to control the bleeding, but it was possible. It was necessary to apply clamps across the aorta above and below the hole in the aorta and to apply a side-clamp to the pulmonary artery.

When that was done, the bleeding was controlled, and it was possible to suture the aorta and the pulmonary artery.

In all the other patients I have used that technique which was forced upon me the first time in this case.

Now we have somewhere between thirty and thirty-five successful cases in which we have used the cross-clamping on the pulmonary artery.

It is possible to excise all the ductus tissue out of the aortic wall, and get normal aortic wall edges sutured against each other, which may be more safe than to leave tissue behind which might later give difficulty in forming aneurysms. In our whole series,† we have had only one recurrence, and that was in a divided septic patient.

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*See page 305 for article by Jones.

†Seven with sepsis; all the other ones without infection.

This was an infected patient in which I divided the ductus. She seemed to do quite well at first, blood cultures were negative, and she was sent home as cured. Two or three months later she came back to the hospital with the same condition she had had when she first entered. I tried to reoperate, and at operation it was found that a sort of aneurysm had developed between the aorta and the pulmonary artery, and there was a wide communication between the two vessels.

Since then I have used the pericardial flap, which always covers the ductus as a sort of plastic material to suture down in the bottom of the groove between the pulmonary artery and the aorta after division of the ductus.

I quite agree with Dr. Jones that it is advisable to have a very good approach in cases like this. I have used the same incision shown you here today, for the ductus, usually taking away the fifth rib, sometimes the fourth.

If bleeding complications should occur during operation it is essential to have a very wide incision to be able to control a severe bleeding.

We have had in our series, two deaths. The first one was the patient with the recurrent condition, just described. She died on the operative day after we had tried to close her fistula once more. We could not close it at operation, and she expired a few hours after operation.

Another patient who died was a little child, about 5 years of age. She had one of the shortest operative times that we have had, and everything was quite uneventful. When the operation was finished, an aspirating tube was passed through the intratracheal tube, and she was aspirated with too high a negative pressure. I didn't observe that the catheter passed through the intratracheal tube was passed with difficulty, so I believe that the sudden death must have been due to a very rapidly increased negative pressure inside the bronchial tree. She had an acute heart stop. We re-entered and massaged the heart, and were able to get her back to life, but the time it took was too long. She developed cerebral symptoms, and died within twenty-four hours after operation.

I would be a little afraid of tearing and pulling with the type of clamps Dr. Gross showed just now. On these heavy clamps, you do not need to have much movement in order to tear a fragile ductus and have bleeding. It must be necessary to be very careful in using those clamps, and it might be safer to use cross clamping of the aorta and side clamping of the pulmonary artery.

DR. OWEN H. WANGENSTEEN, Minneapolis, Minn.—My associate, Dr. Varco, and I want to record a minor variation in the technique of closure of the patent ductus arteriosus.

All of us are aware of the great impetus Doctor Gross has given thoracic surgery in developing his own interest in vascular sugery. In Minneapolis, there has been an intense interest in the subject of patent ductus for a long time, but we, as our Chairman put it yesterday, had our eye on the sight but we failed to pull the trigger.

In 1935, Dr. George Fahr, well-known Minneapolis internist, had a patient of 22 years with a patent ductus for whom he recommended the operation since done by Dr. Gross. Dr. Elliott Cutler in February of that year came out to Minneapolis to give the Judd Lecture. Dr. Cutler, Dr. Fahr, and I discussed the problem at length and subsequent to Dr. Cutler's visit, he and I exchanged letters upon the matter. Dr. Cutler's parting advice to me upon the matter, as I remember it, was to put a Parham band upon the ductus.

Our patient had a large heart and cardiac decompensation. As I recall it, Dr. Gross's first patient was 7 years of age. The experience that my associates and I have had in operating upon patients past late adolescence suggests that the operation in such individuals is ordinarily more difficult than in the younger age groups. It was providential that Dr. Gross chose for his first trial with the procedure a young child, in whom the problem of dissection was simpler.

Since Dr. Gross's description of division of the ductus, Dr. Varco and I have followed this procedure exclusively. We have had the experience of recanalization following ligature of the ductus, which is not pleasant.

(Slide.) We have now divided the ductus in thirty-three consecutive cases without deaths or recurrence; in fact, we have given up ligature altogether. The special point which we wish to emphasize in the dissection is the development of a tissue plane to facilitate the dissection at its most difficult point, the anteromedial angle between the aorta and the pulmonary artery. The fibrous layer of the pericardium is tougher than the walls of the two adjacent vessels, which the dissection proposes to separate. In consequence, this part of the dissection is always slow and difficult. In some instances, as Dr. Gross has been at pains to point out, the lappet of pericardium over the ductus may be dissected off readily. Many times, however, such is not the case, In those instances, we define the outer, tougher, fibrous pericardium and dissect it off the thin, inner, serous pericardium. Failing to do this readily, we open the pericardial sac deliberately and free the ductus from the outer fibrous layer of the pericardium. In the presence of a large heart and a large pulmonary artery, especially in older children or adults, this dissection is not easy. Nevertheless, we feel, after having employed this scheme routinely in all patients in whom the ductus has been divided, that this expedient simplifies the most difficult part of the dissection. Having dissected off the fibrous pericardium, the separation of the aorta from the pulmonary artery on their medial aspects becomes now an easier dissection. In the final stages of the dissection, employment of an illuminated lucite elevator or dissector has proved very helpful in identifying the diaphanous fascial layer before the aorta and pulmonary artery, after the fibrous pericardium has been dealt with.

In the division of the ductus, we employ three long, straight hemostats. Two are put ordinarily on the pulmonary artery side and one on the aortic side of the ductus. The wider cuff is left on the side with the single clamp. The upper clamp on the pulmonary side is removed and the cuff oversewn, as described by Gross. Before removing the remaining hemostat, however, we place a ''stick-tie'' suture behind the clamp, the suture being anchored in a remnant of fibrous pericardium. This scheme insures satisfactory hemostasis without bleeding. It is important not to place this suture in the arterial wall, lest the lumen of the vessel be entered, thereby inviting troublesome oozing. The isolation of a remnant of fibrous pericardium in juxtaposition to the remaining hemostat, on the pulmonary artery as well as aortic side of the ductus, affords an excellent anchorage for the ''stick-tie'' suture which will insure satisfactory hemostasis.

We have employed this method in the instance of a boy of 20 with a large, broad communication between the pulmonary artery and the aorta in which the two vessels appeared to fuse without the intermediary of a connecting ductus.

DR. ARTHUR S. W. TOUROFF, New York, N. Y.—Since returning from the Army, I have operated upon five patients within a relatively short period of time; and accordingly, assume that there are still many patients suffering from patent ductus arteriosus, who will sooner or later come to surgery. The subject of ductal surgery, therefore, remains timely.

My experience with ductal surgery began early in 1940, when I had occasion to operate upon four patients suffering from patent ductus with superimposed subacute bacterial endarteritis. Some of you may recall that the results were reported at the 1940 Meeting of this Association and that they were not particularly brilliant. Among the four patients, there was one cure of the infection, two deaths from hemorrhage, and one in which operation failed to influence the course of infection.

Subsequently, with increasing knowledge of the problems presented by operating in the presence of infection, the results improved. By the time I entered the Army in 1942, I had operated upon nine additional patients with infected patent ductus. Of these, seven were cured and two unimproved. There were no operative deaths. Today, with the use of massive doses of penicillin, it can be assumed that there will be little necessity for operating in the presence of active infection. However, our results did establish conclusively that surgery alone was curative. I might add that our eight cured patients have been followed for from four to six and one-half years and that all of them have remained well. This, in turn, would indicate that operation tends to protect against the development of infection.

Both Dr. Jones and Dr. Gross have mentioned cases in which they assumed that recanalization of the ductus occurred after ligation. While in some of their cases the patient developed unmistakable evidence of recanalization as evidenced by typical changes in bloodpressure, in others the only evidence on which the diagnosis was made was the persistence of a murmur. I do not believe that the mere presence of a murmur, without the other criteria of ductal patency, indicates that the ductus is open.

In the first patient with patent ductus complicated by superimposed endarteritis there was serious operative hemorrhage. Fortunately, the hemorrhage was controlled but, in the process, the ductus was doubly clamped, completely divided, and ligated. We had a sterile stethoscope available during operation and listened to the murmur periodically. It was interesting to note that even after the ductus had been divided and ligated, a loud murmur persisted over the pulmonary artery. When the pulmonary artery was compressed, the murmur disappeared only to reappear when the pressure was released. I assumed that the murmur was produced by the swirl of blood through the dilated pulmonary artery. An analogous situation obtains in cases of congenital dilatation of the pulmonary artery. In such cases, a characteristic murmur often is present. In the case which I have cited, the murmur persisted for several months despite the fact that the circulatory status became normal promptly. I have encountered similar situations on a number of occasions since then and, therefore, am not inclined to accept a diagnosis of postoperative ductal patency merely on the presence of a persistent murmur.

In those instances in which a ductus actually is patent following operation, patency may be attributed to incomplete occlusion by the ligature at the time of operation or to sloughing secondary to infection following operation. Both Dr. Jones and Dr. Gross have stated that in a number of their cases they tied their ligatures rather lightly for fear of cutting through the ductus. Furthermore, I believe that Dr. Gross originally recommended the use of cellophane as a ligature material because he feared a tight silk ligature and deliberately sought some material which would produce a substantial degree of periductal fibrosis and thereby result in gradual complete occlusion of the ductus. I, personally, have tied two or three heavy silk ligatures about the ductus as tightly as possible. In none of the cases has there been evidence of cutting through of the ligatures following operation, despite the fact that a great deal of force was applied in tying them.

Assuming that a ductal ligature has not been tied loosely, it is my impression that recanalization is the result of cutting through, induced not by tying the ductus too tightly, but by infection. Furthermore, I believe that secondary infection also is responsible for late hemorrhage such as has been reported by Dr. Jones. Although Dr. Jones's patients demonstrated no clinical evidence of infection, it seems fair to assume that the granulation tissue and the pulmonary erosion which were found at the time of reoperation could only have been the result of continued low-grade infection. In view of the time interval which had elapsed between the first and second operations, one would expect to find firm scar tissue everywhere in the local operative area. Instead, Dr. Jones found soft granulation tissue and a local erosion of the lung, although cultures proved sterile. I would like to ask Dr. Jones whether the patient received penicillin or sulfonamides, and if so whether they might not have been responsible for the sterile cultures?

In regard to ductal division, I have had experience in only three cases. There were all early in my experience with ductal surgery. None of them was planned and all of them were done in an effort to control accidental hemorrhage. Although first to perform ductal division, you now can appreciate why I have long considered it a doubtful honor. While overseas, I received from Dr. Gross a reprint of the article in which he first described his technique of planned ductal division. In view of the operative findings previously encountered in certain cases, notably adults, I wrote him that I would be very much interested in hearing of his future experiences with the operation. In operating upon adults, I often found the ductus to be sclerotic, thinned out, and sometimes calcific. The pulmonary artery usually was much more dilated and thinner than in children, apparently because of the fact that the lesion had been of longer duration. Not infrequently, the sclerotic ductus was firmly adherent to the thinned-out, dilated, pulmonary artery. I found that such a state of affairs was much more likely to be attended by accidental operative hemorrhage than in cases in which the ductus was soft and rubbery and had much more nearly the consistency of a normal vessel.

I would like to ask Dr. Gross whether he has been able to divide the ductus as safely in adults as in children and whether he has specifically applied the method in cases in which the ductus was obviously sclerotic and rigid. On theoretical grounds, it would appear to be more hazardous because of the poorer tendency of the rigid-walled ductal ends to be apposed by sutures and of healing to take place. However, Dr. Gross's experience may indicate that the danger of ductal division in adults is more theoretical than real.

In closing, I would like to say a few words in regard to ductal division in infected patients. Dr. Crafoord referred to a case in which he attempted to divide an infected ductus, and regretted it. His experience would tend to support my contention that, in the presence of infection, healing is poorer and complications apt to occur. In the presence of infection, it therefore appears wise to avoid ductal division if possible. If division should become necessary as a result of hemorrhage, I would recommend the avoidance of any operative technique which requires the introduction of a considerable amount of suture material, and the use of two, simple, fairly heavy silk ligatures which are placed as far back on the ductal ends as possible. In the three cases in which I had occasion to perform ductal division in the presence of infection, this method was used and was followed by uneventful recovery.

DR. EDGAR W. DAVIS, Washington, D. C.—I wish to call your attention specifically to one patient, a 17-year-old boy with a moderately enlarged heart upon whom surgery was performed. The duct was found to be about 8 mm. long and only about 6 mm. in diameter. Two ligatures of cotton tape were easily applied around the duct and were tied tightly. About 50 per cent of the murmur persisted.

Dr. Touroff's original report described the persistence of a murmur in one patient in which the duct had been divided. Dr. Crafoord has just stated that in one of his patients, in whom the duct was divided, the murmur persisted. In Dr. Blalock's series, the only persistent murmur was in one in which the duct had been divided. These reports, therefore, make one wonder if there is possibly some other source of the murmur. With this in mind, I advised another operation for the patient mentioned above.

Three weeks following the initial surgery, the second operation was performed. The ductus arteriosus was surprisingly short and of such small diameter that it was almost inconceivable that it could be patent. In fact, the duct was found with difficulty and then only after finding the knot of ligature which had been applied at the first operation. The duct was then isolated and two more ligatures of umbilical tape were applied, one on top of the other, because the duct was too short to apply them side by side. Ten days later, when the patient was discharged, a murmur was not heard. Six weeks later, when he returned for a checkup, a murmur was present. Although the patient has a persistent murmur, he is clinically cured, as evidenced by the normal size of the heart and his ability to engage in competitive sports. I doubt seriously that this duct, with four ligatures around it, all of them tied tightly, could be patent.

Our series consists of twenty-six operations performed upon twenty-five patients. Our youngest patient was 22 months old and the oldest was 44 years of age. Both infected and noninfected patients are included. Complications consist of temporary hoarseness in one instance which cleared in a week, and the aspiration of bloody fluid from the first three patients. Since then, no thoracenteses have been done. The mediastinal pleura has been closed in the last three patients. No deaths attributable to surgery have occurred in this series. One death occurred ten weeks after operation, from intravenous heparin and penicillin given continuously. The family physician suspected subacute bacterial endarteritis, but could not prove the diagnosis. No murmur was present just prior to death. An autopsy was refused. With the exception of the patient who was operated upon twice, none had either a persistent or a recurrent murmur.

After we had used double ligation in fourteen cases of patent ductus arteriosus, we decided to divide and suture in the next fourteen patients and then compare the results of the two methods. However, when we studied our first fourteen cases and found that we had such good results, we did not feel justified in changing from a very simple procedure to one which is obviously hazardous and technically much more difficult.

I am not attempting to be a "balance wheel," neither am I opposed to the "division and suture" method as advocated and so ably used by Dr. Gross. If, in subsequent years, we find that the division and suture method can be done with the same degree of safety, with the same or lower mortality rate, I am all for it. However, until then, those of us who perhaps are not the masters of vascular surgery, as Dr. Blalock, Dr. Crafoord, Dr. Gross, and a few others, will find it a much safer procedure to do a double ligation with No. 7 cotton tape, than to divide and suture.

DR. JOHN JONES, Los Angeles, Calif.—Our severest critics, the cardiologists, use two criteria for establishing a diagnosis of the recurrence of the murmur. One is that when they hear a continuous murmur over the pulmonic area, they assume that the fistula has either reestablished or that the ductus was never closed off completely at the time of ligation. The other is the sound track. Those two we have depended upon and done routinely, and, if we detect a continuous murmur in the sound track, we assume that the fistula has re-established or the fistula was never closed off.

The remark about having proved that dilated pulmonary artery makes me feel better. Perhaps some of mine that recurred were that, but we must go on the assumption when we hear the murmur when we take the dressings off a day, or two, or three, or a week after operation, that probably the ligation wasn't tight enough.

In the case that you mention, Dr. Touroff, about there having been a possibility of infection at the site of ligation that caused the fistula between the aorta and the lung, the material was examined by the pathologist, and he reported fibrous tissue, blood clot in various cases of organization formed by reaction, and no evidence of inflammatory reaction in this tissue removed; so I must assume that it was not an infection and was formed by the reaction and erosion by the tapes.

If we had had an inflammatory reaction or an infection at the site of the ligation, we would have had the same experience as in an early case reported in Los Angeles, where we had an endarteritis, and vegetation was both on the pulmonary side and the aortic side of the vessels in autopsy.

DR. ROBERT E. GROSS, Boston, Mass.—Dr. Touroff asked about the ages of patients and the question of performing divisions in adults. I should have mentioned that our patients varied from 2 years to 46. I would have been hesitant at first about undertaking divisions in adults, but I think that if you gain experience in youngsters, where it is easy, you can carry out the procedure in adults.

Regarding the question of division in infected patients, we have operated upon eleven infected patients. Three came within the period in which we have been doing divisions, and with some trepidation division was undertaken. In no instance was there any bleeding or any untoward difficulty. I did, however, take the precaution of placing something between the aorta and the pulmonary artery. This was a flat piece of pectoral muscle fascia, about 2 cm. square, which was interposed between the aortic and pulmonary artery suture lines. Dr. Wangensteen indicated that pericardium can be pulled between the suture lines. This seems to me to be an admirable suggestion.

LUNG RESECTION FOR PULMONARY TUBERCULOSIS

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PHILADELPHIA, PA.

PULMONARY resection is a relatively new type of treatment for tuberculosis, at least in its modern form. As yet it is under some cloud because of the dramatically poor results first obtained. However, most new procedures, methods, and mechanisms have certain inherent bad features which can be eliminated only by persistence and increasing experience. After an experience with eighty pulmonary resections for tuberculosis, I feel satisfied that the procedure is here to stay, will become progressively standardized, and will become increasingly more satisfactory as regards long-range "cures."

First, it is painfully apparent to every phthisiologist that collapse therapy is unavailing or actually dangerous in certain types of tuberculous involvement such as tuberculoma, stenosis of large bronchi with definite retention of secretions, and persistent check-valve or tension cavities, and should never be utilized in them. To this list might be added cases of unilateral, caseous pneumonia with extensive cavitation throughout an entire lung (the "destroyed lung"). In addition, there are many cases in which collapse therapy has already failed repeatedly, or in which it will probably fail. These are cases in which satisfactory anatomical collapse by thoracoplasty has failed to convert the sputum (the same with pneumothorax) and certain cases of lower lobe cavitation, giant cavities, and residual tuberculous bronchiectasis.

It is evident that there is a very definite need for some effective type of treatment in such cases. Pulmonary resection would seem to be the answer, providing it can be done with an acceptable mortality and morbidity. It must be noted that this mortality and morbidity does not have to be comparable to that in thoracoplasty or pneumothorax therapy, since resection is recommended only in cases unsuitable for these treatments. Of course, if and when the mortality and morbidity of resection is reduced to levels comparable to that of collapse therapy, then some consideration may be given to generally replacing collapse measures by extirpation of the patient's most malignant tuberculous focus.

It is essential to remember that tuberculosis is always a generalized disease, with demonstrable foci in lungs, kidneys, spleen, bones. One must not forget that we can only hope to extirpate or heal the most malignant tuberculous lesion, whether it be by lobectomy, thoracoplasty, nephrectomy, arthrodesis, spine fusion, or amputation of an extremity. We trust that the patient will then be able to heal all other foci in his body. Hence, he still must be under

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a general therapeutic regime for a prolonged period of time. It must be fully recognized that if the patient is suffering from a progressive generalized or even multiple focus tuberculosis, our local efforts are doomed to eventual failure except in rare instances. Thus pneumonectomy for acute caseous pneumonic disease bears a much poorer eventual prognosis than pneumonectomy for severe bronchial stenosis with essentially healed parenchymal disease, since in the former case the generalized disease process is much more likely to produce another virulent focus in the other lung or elsewhere. This consideration does not necessarily contraindicate pneumonectomy in acute pneumonic disease, but *it does indicate that the eventual prognosis and statistical results in such cases* must inevitably be poor.

Having mentioned the generally recognized indications for pulmonary resection in tuberculosis, and having suggested the possible results obtainable by technically successful operations, we must consider in some detail the methods by which technical success may be routinely obtained.

The commonest causes of mortality and morbidity in resection for tuberculosis are: (1) asphyxia during the operation due to mechanical expression of copious secretions into the bronchial tree of the nonaffected lung; (2) spread of the disease to the contralateral lung or to the remaining portion (if any) of the same lung; (3) hemorrhage or shock; (4) postoperative opening of the bronchial stump, producing a bronchopleural fistula; (5) tuberculous empyema, early or late; (6) pyogenic empyema; (7) a combination of the last three complications; and (8) marked departures in postoperative pleural pressures from the normal.

Let us take up each consideration in detail.

1 and 2. Prevention of obstruction of the airways by copious secretions from the affected lung is extremely important. In the usual lateral or posterolateral operative approach, and even the anterolateral approach, gravity insures that any secretions expressed from the diseased lung must pass into the trachea and then enter the bronchial tree of the more dependent lung. The anesthetist may be able to aspirate part of these secretions with a catheter introduced through an endotracheal tube. In cases where the secretions are copious, he must inevitably fail to remove all of them, since the tracheal aspirations must be interrupted by longer periods during which anesthetic gases are introduced. Once these secretions enter the smaller bronchial branches, they can only be removed by coughing, since even the bronchoscopic aspirator passed under direct vision cannot evacuate the contents of obstructed bronchioles. Add to these mechanical factors the reluctance of many anesthetists to aspirate through the endotracheal tube unless audible bubbling of secretions is present, and one will see that such nonchalance in surgical approach and anesthesia must be attended by considerable risk of actual asphyxia, and, where this is avoided, of obstruction of the smaller bronchi by infectious secretions. This latter situation leads in many cases to segmental atelectasis and tuberculous pneumonitis ("spread").

One would think that endobronchial *anesthesia would prevent the development* of asphyxia or contralateral tuberculous spread. Unfortunately, the usual type of endobronchial anesthesia tube is arranged to be passed blindly. Thus, one cannot be completely sure that the tip of the tube is in the appropriate bronchus, or how far in. In an attempt to improve this method, Dr. Henry S. Ruth and myself have modified a Woodbridge-Flagg type of tube so that the obturator is really a 5 mm. bronchoscope. This may be simply and accurately

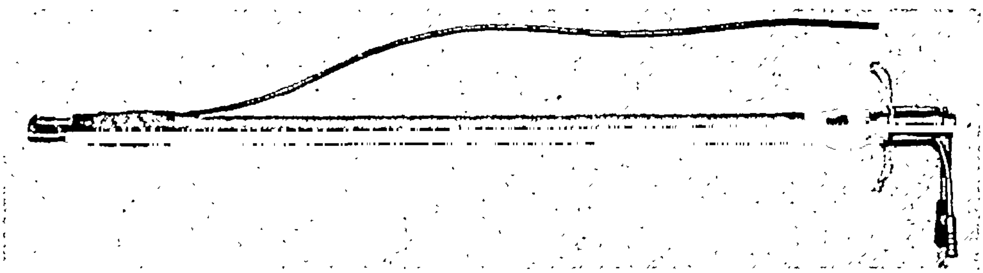


Fig. 1.—Direct vision endobronchial catheter, obturator in place. The rubber sheath has been drawn back from the tip to show the coil-spring construction. Note inflatable cuff. Obtainable from Richard Foregger Co., New York City.

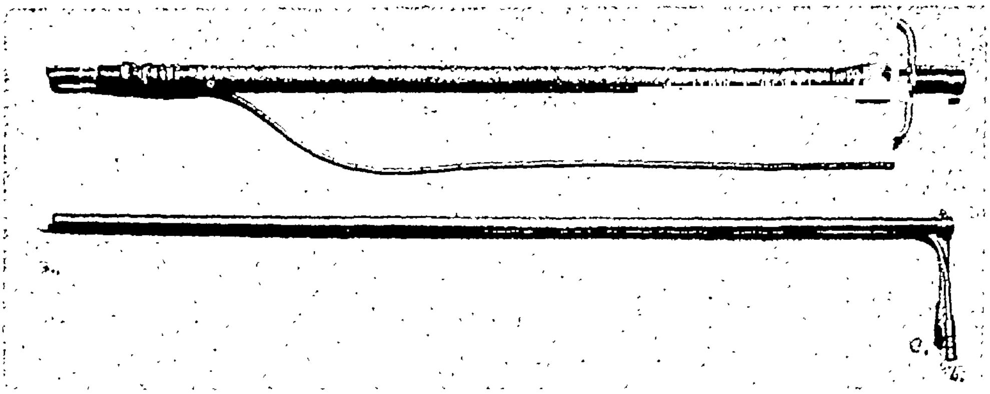


Fig. 2.—Obturator removed. It is really a 5-mm. bronchoscope with a built-in light carrier (see bulb-a) and oxygen delivery tube (b). Battery cord is connected to terminal (c).

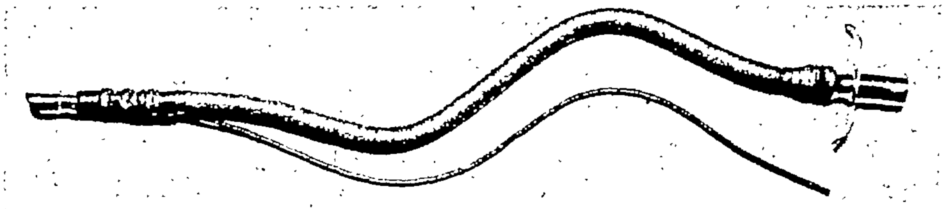


Fig. 3.—Flexibility of tube after removing obturator. This lessens trauma to air passages.

passed into the bronchus of the normal lung for the desired depth. An inflatable rubber cuff close to the tip obstructs passage of air or mucus between the trachea and the intubated bronchial tree. (See Figs. 1, 2, and 3.) A very satisfactory endobronchial anesthesia is thus obtainable and contralateral spread or asphyxia from secretions would seem to be preventable by this method. However, sometimes the inflatable cuff may become deflated. Therefore, it is advisable to employ a considerable degree of Trendelenburg position, so that gravity will aid in keeping secretions from passing by the possibly deflated cuff. Another factor in this type of anesthesia is the frequency with which the right upper lobe bronchus is obstructed by the inflated cuff. This permits patients undergoing left-sided surgery only the right middle and lower lobes for oxygenation of their blood. In the hands of a good anesthetist, no difficulty will be observed during such anesthesia, but considerable aid by manual compression of the anesthesia bag may be necessary.

In cases of lobectomy or partial lobectomy, endobronchial anesthesia offers no protection against homolateral spread of secretions, and it prevents periodic inflation of the lung being operated on, which may be important, especially in segmental surgery.

For these reasons, even having a satisfactory endobronchial tube, we use it only in cases of total pneumonectomy and lower lobe lobectomy. In upper lobe resection, middle lobe resection, and those total pneumonectomies which we expect to be technically easy, a modification of the Rienhoff anterolateral approach is used with the patient in an extreme Trendelenburg position on the operating table. The approach is really an anteromedial one through the third anterior interspace with or without resection of an adjacent rib. The good lung is elevated above the diseased one by a pillow under the shoulder of the unaffected side, or by tilting the operating table laterally. For such cases, catheter suction through a simple endotracheal tube provides efficient evacuation of secretions which either stay in the affected lung or lobe, or, if they do enter the trachea, do not cross to enter the upper lung.

The Overholt support, which permits a posterolateral type of operative approach with the patient in a slight Trendelenburg and the face down position, the disease (operative) side being tilted lower than the good side, appears to overcome most of these problems. In this position, a very large surgical exposure is obtained, while secretions are expressed out of the diseased lung only with difficulty, and then drain into the trachea and out the mouth by gravity. Gravity is assisted by suction through a simple endotracheal tube. Unfortunately, this method, too, does not completely prevent homolateral expression of secretions (homolateral atelectasis and spread).

3. Hemorrhage and shock in resection for tuberculosis does not differ from that seen in resection for other pulmonary diseases. It may be combated in the usual way by gentleness, meticulous hemostasis, infusions, and transfusions. The average pneumonectomy or lobectomy patient probably loses 1,500 c.c. of blood during the operation and the next twenty-four hours. This should be replaced while the patient is on the operating table or soon after. Some patients, of course, may lose much more blood.

4. Opening of the bronchial stump is now, fortunately, becoming rather rare. Almost every thoracic surgeon has his own method of bronchial closure and every one is getting improved results. Essentially, most thoracic surgeons have stopped expecting the bronchus to heal *per primam*. They merely close it with fine sutures which are placed so as to interfere as little as possible with

7. Opening of a bronchus into the pleural cavity ordinarily leads to a mixed tuberculous empyema, although either pure tuberculous or pure pyogenic infection may sometimes ensue. Either of them appearing primarily may predispose to opening the bronchial stump. This serious combination of complications is frequently fatal and requires rigorous management. While the routine employment of a thoracotomy, thoracoplasty, and finally, Schede type of program, may eventually salvage the patient's life, it is a prolonged and uncertain course. Perhaps we should boldly invade the tuberculous pleural space, and reamputate and reclose the bronchus, even if this should mean further pulmonary resection. Fortunately, such complications become rarer as time goes on, and may eventually become practically nonexistent.

8. Morbidity or death from pleural pressure changes may be related either to air leaks from the sutured bronchus or lung tissue, to pleural effusion or bleeding, or to homolateral shifting of the mediastinum due to excessive escape of pleural air or fluid from the operative pleural rent, which can seldom be closed in a truly airtight manner. (See Fig. 5.) In order to control these possible departures from the normal, most thoracic surgeons resort to frequent thoracentesis and to determination of the plueral pressure levels in the tightly closed chest. Some surgeons routinely drain lobectomy cases but depend upon thoracentesis to control pleural pressures in the total pneumonectomies. Various experiences, some very dramatic, have led the author to adopt tne policy of drainage in every case at a constantly maintained negative pressure (-15 cm.). In pneumonectomy patients, the drainage tube is removed in forty-eight hours. In lobectomy patients, a suction motor is used to exaggerate the negative pressure somewhat, and the tube is removed in three to four days, when postoperative x-rays have revealed complete lung re-expansion. (See Fig. 6.)

Besides the adequate management of each of these factors, there are certain other very important considerations which must be satisfied if routinely good results are to be obtained. First, the surgical technique must be meticulous. Individual ligation of the hilar vessels and careful closure of the bronchus are so essential that it may be said that a lung tourniquet should be on the operating table only to protect against an extreme emergency. It has no other use in pulmonary resection for tuberculosis.

Postoperatively, these patients should ordinarily be bronchoscoped before leaving the surgical theatre. Subsequently, great pains should be taken to maintain complete evacuation of the air passages. The cough mechanism is greatly impaired by the presence of a paralyzed diaphragm even after a total pneumonectomy. This is due to unstabilization of the mediastinum because of the atonicity and paradoxical movement of the operated diaphragm. Thus, in the inspiratory phase of the cough cycle (inspiration, compression, and forceful expulsion of air) the paralyzed diaphragm is drawn up into the thorax, permitting the mediastinum to be drawn toward the unoperated side. Thus, a full inspiratory filling of the remaining lung is not attained.

Similarly, diminishing the rigidity of the chest wall by extensive or multiple rib resection is attended by impairment of expectoration because of interference

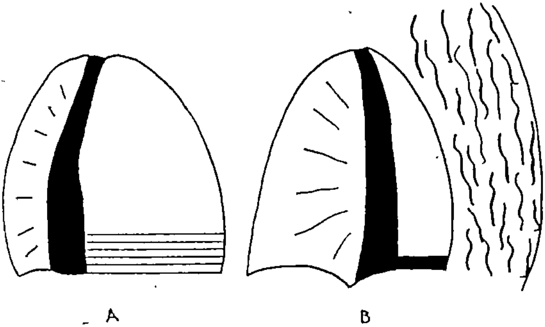


Fig. 5.—*A*, After pneumonectomy the heart and mediastinum may be forced toward the opposite side due to leakage of air or accumulation of fluid under pressure in the pleural space. *B*, After pneumonectomy air and fluid may escape during coughing from the pleural cavity through a pleural rent into the layers of the chest wall producing emphysema or ecchymosis. The very high negative pressure produced in the pleural cavity draws the mediastinum toward it causing torsion of the great vessels and overdistension of the remaining lung. Simple water-seal drainage may produce a similar high negative intrapleural pressure.

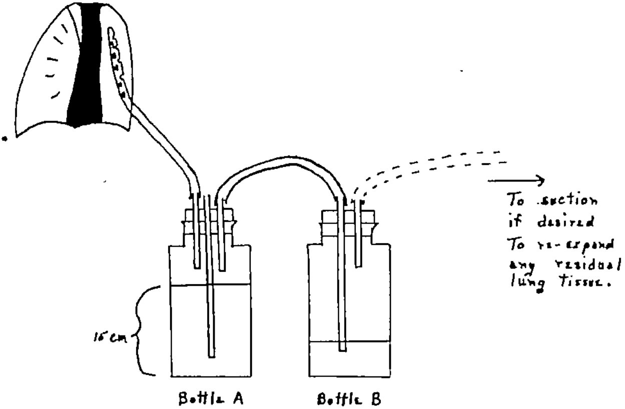


Fig. 6.—Constant pressure pleural drainage system. Bottle A prevents pleural pressure from becoming more negative than −15 cm. water (or whatever the height of the fluid column is). Bottle B is a simple water-seal trap to prevent positive pleural pressure from developing. To re-expand any residual lung tissue a mechanical sump pump is recommended—attach to small tube of Bottle B.

with both the inspiratory and compressive phases. Pain, too, both by reflex and by voluntary limitation of costal excursions tends to inhibit deep breathing and free coughing. The author, therefore, never intentionally performs a phrenic paralysis during a pulmonary resection except in those instances where rupture of a tuberculous cavity demands extreme efforts at obliteration of the contaminated pleural space. Phrenic exeresis or phraxis may be performed in a week or two after full respiratory compensation has taken place. Likewise, no more than one rib is removed for entrance into the thorax, and usually none when the anterior approach is used. Careful closure of the pleural rent is always attempted. In order to lessen postoperative pain, the intercostal nerves of the rib above and below the incision are picked up with a hemostat and crushed before closing the chest. Thus, a relatively painless postoperative course is obtained.

Even with the foregoing measures, there are some persons who cannot effectively cough subsequent to these operations. If turning from side to side, sitting the patient up in bed, and the like, prove ineffective, transnasal tracheal suction should be employed by the method perfected by Dr. Lyman Brewer. A relatively new, large-caliber Levine tube is passed through the largest nostril into the pharynx. The tongue is pulled forward and the patient is encouraged to pant vigorously. On an inspiration the tube is passed further, the tip of it tending to enter the glottic aperture. The patient immediately chokes and gasps and is unable to phonate. A strong suction motor is now connected to the Levine tube, which is moved back and forth in the trachea, passing even into the lower lobe bronchi on one or both sides. If the patient becomes cyanotic the suction may be stopped temporarily with the tube in place. The violent bronchial peristalsis induced by this procedure throws much mucus out of the bronchioles into the larger bronchi and trachea, where it is either aspirated or easily coughed up after the tube is removed. This procedure may be performed by the nursing staff at two- to four-hour intervals.

For patients in whom the tube can be passed only with difficulty or where sudden rise of temperature and x-ray evidence of "spread" (really atelectasis first) are observed, formal bronchoscopy should, of course, be done.

In persons requiring frequent tracheal aspirations for copious secretions where there is difficulty in transnasal aspirations, a formal tracheotomy may be done. Then the aspirations may be carried out as often as every fifteen minutes by a catheter inserted through the tracheotomy tube. Between periods of aspiration, oxygen may be administered directly into the trachea by a small catheter passed through the tracheotomy tube. Such oxygen flow should seldom be over 3 L. a minute, and the oxygen should be moistened by bubbling it through water before it reaches the catheter. No difficulty in healing of the tracheotomy sinus has yet been observed.

The patient should be got out of bed as soon as he is able, perhaps by the first postoperative day. This seems to improve both expectoration and the general condition.

Subsequent to an initial successful pulmonary resection, the question of thoracoplasty or phrenic nerve paralysis always comes up. In our series the treatment has varied with increasing experience. While at first no thoracoplasty nor phrenic paralysis was performed except in the treatment of a complication such as an empyema, today one or the other procedure is done as a routine. Thus, a temporary phrenic paralysis is always performed subsequent to a lower lobectomy. A permanent phrenic paralysis and a seven- or eight-rib thoracoplasty are routinely performed after total pneumonectomy. A partial (four- or five-rib) thoracoplasty is performed after an upper lobectomy.

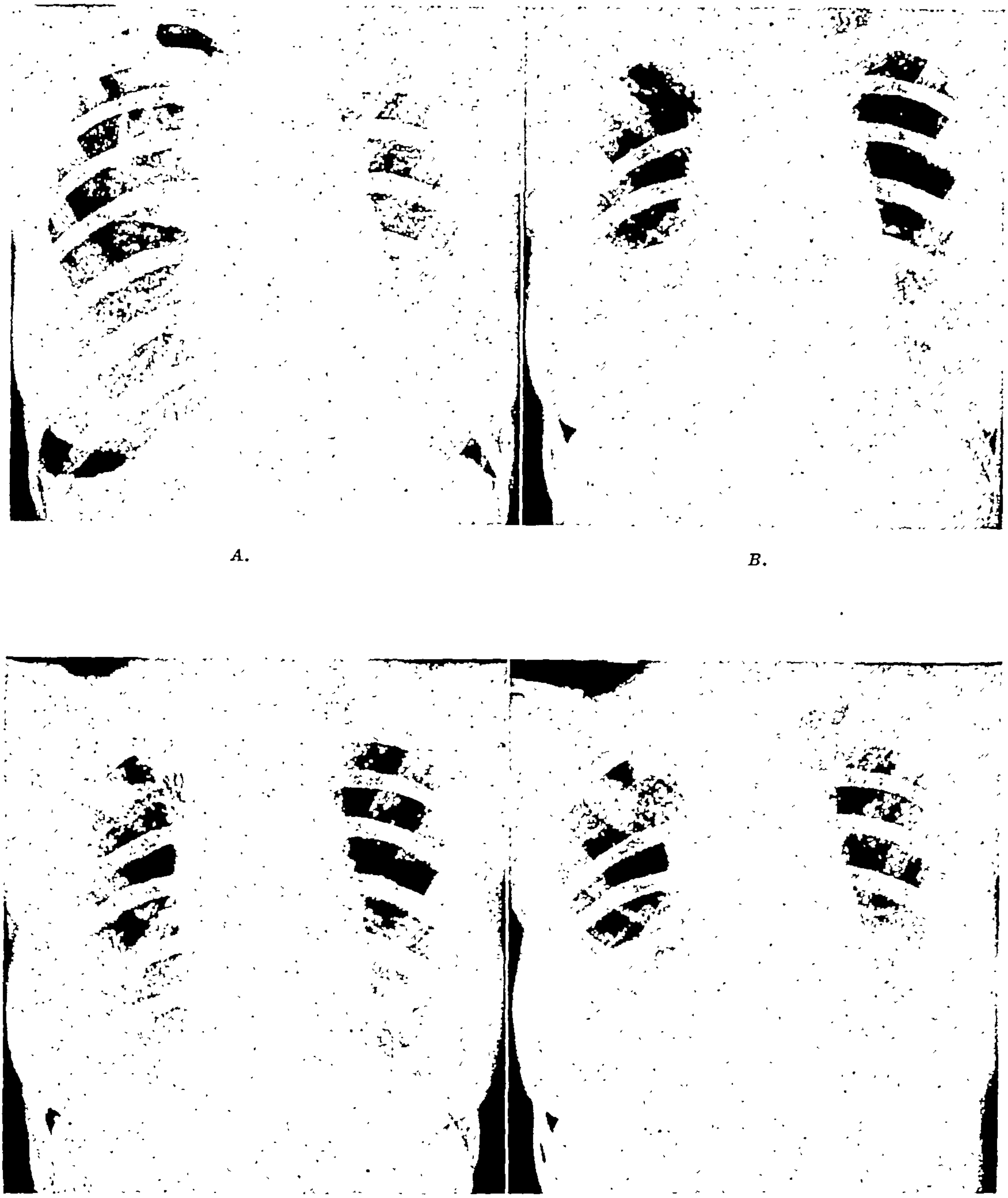
Previously in this paper it has been advised that the pleural space be obliterated quickly and completely by re-expansion of any remaining lung tissue to lessen the risk of tuberculous empyema, and by thoracoplasty where re-expansion obliteration cannot be accomplished. There are two other reasons for reducing the size of the affected hemithorax which to the author seem sound. One is the risk of overexpansion of all remaining lung tissue (on both sides) producing progressive and irreversible destructive emphysema. Much experimental and a growing amount of clinical evidence is available to support this view. The third reason is the presumed risk of breaking down a residual caseous focus within the remaining lung tissue by overstretching this section. Certain of our cases seemed to give presumptive evidence that such breakdown might occur. A huge (giant) cavity developed suddenly in the apex of the residual right lower lobe eleven months after upper and middle lobectomy. This ballooned up so quickly and yet responded so readily to a seven-rib thoracoplasty that the physicians in charge find it hard to believe that the ordinary rules of cavitary formation and closure were applicable in this case. And reasoning backward, if this huge cavity could be controlled after its formation by a seven-rib thoracoplasty, could it not have been more easily prevented by a five-rib operation? (See Fig. 7.)

Naturally, in segmental pneumonectomy the lung is only slightly reduced in size, requiring no thoracoplasty.

As to choice of operative procedure, it is obvious that the entire malignant tuberculous focus must be removed if possible; therefore, pneumonectomy will sometimes be necessary. However, if the process is localized in one lobe, it seems unnecessary to remove the other lobes which may contain small nodules. After all, the lobes in the other lung may also contain such nodules, and so may many other organs such as spleen or bones.

Segmental resection has been performed seven times in this series, twice as part of a combined procedure (another lobe being removed at the same time); and five times as an individual procedure. In general, the course of these cases *was comparable to that in simple lobectomy. After all, the individual broncho*pulmonary segments are functionally as separate as individual lobes, and are not inseparable anatomically.

The indications for this surgery, the procedures performed, and the fatalities are listed in Table I. Four of the cases listed as serious bronchial stenosis were also cases in which thoracoplasty had been previously performed but were not



A. *B.*

C. *D.*

Fig. 7.—*A, B, C, D, E* show development of a large cavity in the overdistended apex of the right lower lobe eleven months after upper and middle lobe lobectomy for tuberculosis. It was readily closed by a seven-rib thoracoplasty. (For Fig. 7, *E,* see opposite page.)

included in the post-thoracoplasty cases. The fatalities and complications listed in Tables I and II are those which have occurred to date. Not all deaths were operative, nor were all due to tuberculosis. An evaluation of the present status of the surviving patients is given in Table III.

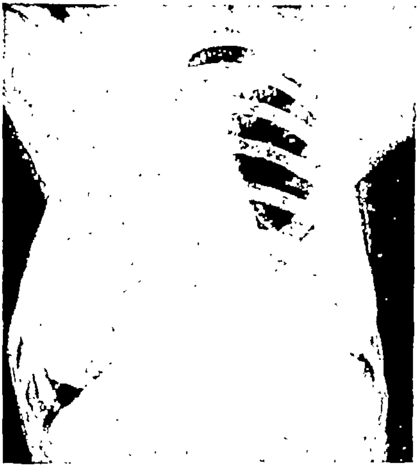


Fig. 7E.

TABLE I

INDICATIONS	CASES	PNEUMONECTOMIES		LOBECTOMIES		SEGMENTAL		% DEAD
		TOTAL	DEAD	TOTAL	DEAD	TOTAL	DEAD	
Serious bronchial stenosis*	9	9	2	–	–	–	–	22.2
Associated or residual bronchiectasis	8	4	0	4	0	–	–	0
Ineffective temporary collapse	4	2	1	2	0	–	–	25.0
Very large cavity	12	5	1	6	2	1	0	25.0
Check valve cavity	3	0	0	2	0	1	0	0
Ineffective permanent collapse	21	9	4	9	2	3	0	28.6
Acute progressive cavitary T.B. (destroyed lungs)	10	9	6	1	1	–	–	70.0
Lower lobe cavity	4	1	0	3	2	–	–	50.0
Elective	7	1	0	5	1	2	0	14.3
Tuberculoma	1	1	1	–	–	–	–	100.0
Total	80	41	15 (36.5%)	32	8 (25%)	7	0	27.5

*Four patients listed as serious bronchial stenosis also had had previous thoracoplasty, but were not so listed.

SUMMARY

Eighty patients with pulmonary tuberculosis have been subjected to pulmonary resection during a four-year period. The indications are the usual ones accepted by surgeons doing pulmonary resection for this disease. The methods

TABLE II. COMPLICATIONS IN THE ENTIRE SERIES TO DATE

COMPLICATION	TOTAL	FATALITIES	PER CENT
Spreads, contralateral	13	6	46.1
Spreads, homolateral	2	0	0
Empyema, tuberculous	5	2	40
Empyema, pyogenic	4	0	0
Empyema, mixed	1	1	100
Bronchopleural fistula plus empyema	7	3	43
Postoperative cardiac failure including pulmonary edema	2	2	100
Operative cardiac arrest, asyphyxial	1	1	100
Due to vagal reflex ?	1	0	0
Chest wall infections and sinuses	6	0	0
Contralateral pleural effusion	2	0	0
Tuberculous meningitis (6 mo. postop.)	1	1	100
Tuberculous pericarditis	1	1	100
Uremia due to amyloid disease (preop.) 3 mo. after operation	1	1	100
Uremia due to transfusion reaction	1	1	100
Acute postoperative anuria	1	0	0
Hemorrhage from a pulmonary vessel (3 days post-tourniquet pneumonectomy)	1	1	100
Slow re-expansion of residual lobes	2	1	0
Late development of bronchopleural fistula	1	0	0
Late development of bronchoesophagopleural fistula	1	1	100
Late re-exacerbation of tuberculosis	2	1	50
Total	56	23	

TABLE III. BREAKDOWN OF PRESENT STATUS OF LIVING PATIENTS

PRESENT STATUS	NO.	%
Satisfactory with negative sputum	39	68.4
Sputum-positive (including the bilateral cases)	6	10.5
Empyema	3	5.2
Draining sinus	5	8.7
Bilateral (preop.)	3	5.2
Spread with progression	3	5.2
Spread without progression	4	7.0

and details of management which have lessened the formidability of the procedure in our hands are given in some detail. The results and complications to date are listed. They seem to indicate in a general way the reasonable expectation of success which may be entertained in the various types of pulmonary tuberculosis.

CONCLUSIONS

Pulmonary resection for tuberculosis is a practical method of removing pathologic foci, which resist collapse measures. The actual morbidity associated with these procedures cannot be accurately estimated at the present time, since our earliest cases were operated on and managed by antiquated and deficient methods, and since very few cases were operated on even five years ago (none in this series over four years ago and many were operated on recently). Since operative complications may not cause death for months or years, the actual operative mortality is difficult to estimate. Statistics of different clinics vary considerably because some physicians may call a contralateral flare-up a spread, while others call it autotuberculinization. Furthermore, one series may include a considerable proportion of patients with a very poor prognosis, such as acute caseous pneumonia, while another may be largely composed of patients with a good prognosis, such as bronchial stenosis or residual bronchiectasis. In still another series they may be largely of post-thoracoplasty patients. Here the operative trauma and shock may be exaggerated by the distorted anatomy, and a considerable risk of operative cavitary rupture is ever present.

But when all this is allowed for, it must be accepted that pulmonary resection is applicable in many cases where no other method of treatment is feasible, and in such patients it presents an operative mortality of probably less than 20 per cent. Further efforts and the elapse of several years will be necessary before exact evaluation is possible.

INTRABRONCHIAL HEMORRHAGE IN BATTLE CASUALTIES

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INTRODUCTION

IN THE Sicilian and Western European Campaigns, 118 men having thoracic wounds were treated by the writer and other members of an auxiliary surgical team. The group included thirty-three with thoracoabdominal wounds and eighty-five with penetrating or perforating thoracic wounds (Table I). Most of these were the more severe "nontransportable" casualties as seen in Field Hospitals. A clinical triad, intrabronchial hemorrhage, ineffectual cough, and anoxia, was of frequent occurrence. In these patients, prompt relief of the bronchial obstruction was urgently required. This report considers the etiological factors, the clinical features, and the method of treatment that was evolved in the management of the respiratory embarrassment in this group. Emphasis is placed on the necessity for immediate and thorough clearing of the tracheobronchial tree as a part of resuscitation. The advantages of bronchoscopic aspiration to accomplish this objective is stressed. A technique of instrumentation without anesthesia, with simultaneous administration of oxygen, and without moving the patient from a semisitting position is advocated. The method proposed was generally used by anesthetists and surgeons of the Third Auxiliary Surgical Group, the members of which performed over 19,000 operations under field conditions in Western Europe, among which 1,306 bronchoscopic aspirations are recorded.

The intrabronchial hemorrhage in the patients to be described was never severe. Impairment of cough was always present, however, and most of the patients having severe symptoms had not been able effectively to clear their air passages for a period of from six to twelve hours. Invariably, those presenting the more severe degrees of anoxia were those in which ineffectual cough had resulted in a "spill" of blood and secretions into the unaffected lung. The factors that were noted to impair cough were, in order of their importance:

1. Dyspnea due to hemothorax and bronchial obstruction
2. Pain and the fear of pain caused by coughing
3. Shock
4. Sucking wounds
5. Lacerations of the diaphragm
6. Cerebral anoxia
7. Morphine narcosis.

It seems likely that in past wars those sustaining thoracic wounds productive of severe respiratory embarrassment have either been included among

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Read (by title) at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

TABLE I. THORACIC WOUNDS

TYPE	NUMBER	NO. DEATHS	% MORTALITY
Penetrating	35	1	2.8
Sucking	50	7	14
Thoracoabdominal	33	6	18.1
Totals	118	14	11.8

the high percentage of chest wounds that are fatal on the battlefield[1] or the men have not survived to reach a hospital. The frequency with which casualties of this type were seen in the recent conflict was doubtless due, in part, to the proximity of hospitals to the front. No less important, however, was the prompt treatment received for sucking wounds and shock in aid stations and the reduction in the time required for evacuation.

BIBLIOGRAPHY

For reasons considered above, the subject under consideration has received scant attention in all except the most recent literature of military surgery.

Jolly[2] writing of his experience with chest wounds in the Spanish Civil War, states: "Almost 90 per cent of the total number of cases show hemoptysis, but in those in which the wound was caused by a bullet, the bleeding rapidly diminishes and usually ceases towards the end of the first week." Again, speaking of the operative treatment for laceration of the lung, he says: "Hemoptysis is seldom severe enough to cause alarm. It ceases spontaneously under general treatment, especially repeated injections of morphine." It can be inferred that these patients had comparatively minor wounds and that their cough mechanism was effective. As will be shown later, the operative difficulties encountered in the thoracic operations reported here, were, in the main, those connected with keeping the airways clear of blood. That Jolly experienced the same difficulty can be deduced from his account of the surgical problem involved in the repair of the lacerations of the lung and diaphragm. Regarding this, he says: "The operation must be performed quickly . . . the patient's condition is precarious throughout . . . many of the more gravely wounded men die on the table."

In Bailey's more recent and more comprehensive volume concerned with war surgery, Edwards[3] does not consider this subject. Magill[4] in the same volume, writing on the anesthesia in thoracic injuries considers the problem briefly: "It is essential to maintain a free airway at all times . . . intubation should be reserved for those operations during the course of which it may be necessary to aspirate blood from the trachea and bronchi." He does not suggest bronchoscopy.

Churchill,[5] writing on his observations of chest injuries in the Mediterranean Theater, makes specific but brief mention of the difficulties caused by bloody secretions in the airways, as follows: "The use of bronchoscopic and catheter tracheobronchial aspiration to clear the respiratory passages of bloody secretions has found increasing use." His observation that oxygen therapy is ineffectual without such aspiration has been confirmed many times in this study.

Sampson[6] working in the same theater, appears to be more enthusiastic in the application of bronchoscopy. In a recent letter he wrote: "We agree perfectly about the value of aspiration of the bronchial tree whenever or wherever there is difficulty in raising. As a teaching principle for other teams we have probably done more catheter aspirations, since this is a bit easier to teach. However, there is no doubt that bronchoscopy is more efficient than the catheter. We have been surprised that bronchoscopy can be repeated so often without ill effect."

In more recent communications regarding general hospital experience with thoracic wounds[7, 8] tracheobronchial aspiration is either ignored or given only passing consideration. In general, it may be noted that the application of tracheobronchial aspiration is advocated most warmly by those whose experience was gained with "nontransportable casualties" in field hospitals. In this connection, comparison of the article of Betts[9] with that of Welch and Tuhy[10] on thoracoabdominal wounds is of interest. The former's experience was largely in field hospitals and was presumably similar in every way to that reported here. He strongly advocates tracheobronchial aspiration postoperatively and states: "We believe that all members of any thoracic surgical team, surgeon, assistant surgeon, and anesthetists, should be competent bronchoscopists in order that the benefits of bronchoscopy will always be available." Welch and Tuhy's experience was in an evacuation hospital. They consider tracheobronchial aspiration in neither pre- nor postoperative treatment. Among other postoperative pulmonary complications they record five instances of atelectasis and four of bronchopneumonia in seventy operative cases, an incidence of 12.8 per cent. Betts,* in an experience with thirty-one field hospital patients who were presumably more gravely wounded, records two fatal pulmonary complications, one from "strangulation," and another from lobar pneumonia, an incidence of 6.4 per cent, or exactly half that of the former series.

In a communication from the Mediterranean Theater, Snyder[11] considers the preoperative role of tracheobronchial aspiration in chest wounds. He advocates intercostal nerve block and catheter aspiration, but adds that, "in some instances bronchoscopic aspirations must be used." He recommends routine aspiration through the bronchoscope at the end of the operation.

Beecher[12] in an article of anesthesia practice, from the same theater, opposes the routine use of the bronchoscope in thoracic injuries, but advocates tracheobronchial aspiration. His objection to bronchoscopy at the end of operation because it necessitates deep anesthesia or the application of a local anesthetic is not substantiated in this study. As will be shown later, bronchoscopy can be safely done in these patients without any anesthesia, local or general, and for various reasons omission of any anesthetic is advantageous.

In contrast with the foregoing, specific and detailed consideration of this subject is found in two more recent communications from the Mediterranean Theater which consider casualties of this type under the title of "Wet Lung." In the first, Burford and Burbank[13] report seventy-nine patients "first seen within forty-eight hours after being wounded, the majority of which were seen within twenty-four hours." They postulate the view that "in all wounds of the chest, to a greater or lesser degree, depending upon the type and severity of the lesion, the lung tissue reacts to produce more than its usual amount of interstitial and intraalveolar fluid." They suggest the "existence of reflex bronchial and bronchiolar spasm originating from a painful chest wall or parietal pleura, which would explain some of the phenomena observed." In their treatment, they stress the desirability of "*abolition of pain and physiologic remobilization of the chest wall*" by intercostal injections (novocain nerve block). With this treatment they report that "the cough becomes painless and effective, and evacuation of the bronchopulmonary tree becomes established." In some instances they utilized tracheobronchial catheter aspiration, and state that, "in a few, bronchoscopic aspiration may be necessary."

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*In a personal communication he reports one nonfatal case of atelectasis.

The second communication of Brewer and his associates[14] considered under the heading of "Causative Factors": "Forces leading to the production of secretions and other fluids in abnormal amounts in the respiratory tract" and "conditions preventing adequate removal of the fluids so produced." To support the view that *abnormal amounts of secretions are produced*, they cite the experimental work of Drinker and Warren[15] and make clinical application of the findings of these authors. They do not evaluate the relative importance of these factors. Like Burford and Burbank, they advocate bronchoscopy only after intercostal nerve block or catheter suction has been tried. They state that topical anesthesia for bronchoscopy must be employed "in all except semicomatose or moribund patients." In addition, they advise that 100 per cent oxygen should be administered through the bronchoscope *during this procedure* to all patients who have anoxia. These practical features of treatment will be discussed later.

The observations of these authors that patients in this category may be confused with pulmonary edema has been confirmed in this study. The familiar clinical signs of the latter, together with the frothy, bloody sputum should make the differential diagnosis easy. In the group reported here, pulmonary edema was occasionally seen in patients who had been given large amounts of plasma prior to whole blood transfusion. They were commonly in a state of irreversible shock.

The writer's experience indicates that, in general, ineffectual cough alone is the basis for the symptoms in this type of injury, and that the benefits reported by these authors with novocain injections are due entirely to the increased effectiveness of cough which followed relief of pain. This view is supported by our observation that striking relief which persisted for several hours usually followed thorough bronchoscopic aspiration (Cases 1, 2 and 3). Pulmonary edema excepted, persistence of "wetness" after any treatment short of bronchoscopy, performed as hereinafter described, appears to the writer to be due to incomplete clearing of the tracheobronchial tree. In general, we preferred to use what is admittedly the most effectual method of clearing the airways (bronchoscopy) at once, correct shock, and then proceed, in most instances, with a definitive operation performed under endotracheal anesthesia, at which time ribs were trimmed and lacerations of the chest wall, lung, and diaphragm were repaired. Bronchoscopy was done routinely at the end of operations and, subsequently, cough was usually comparatively painless and effectual.

ETIOLOGICAL CONSIDERATIONS

The wounds producing the clinical triad previously noted varied greatly. They were rarely caused by small-arms fire. More commonly the injury was produced by a high explosive shell fragment. Usually the foreign body was 1 cm. or more in diameter. It was retained in some instances, and in others the wound was of the "through and through" variety. One, and occasionally two, sucking wounds were usually present and single or multiple shattered ribs were the rule. A fairly extensive laceration of the lung was usually present. Laceration of the diaphragm was common. Hemothorax was always observed; hemopneumothorax was somewhat less frequent. A sizeable bronchial fistula permitting a leak of the hemothorax blood into the bronchi was exceptional. Wounds of the latter type doubtless commonly occurred, but those sustaining them probably died before reaching a hospital. The source of the intrabronchial blood, with one exception, appeared to be a slow pulmonary hemorrhage into a small bronchus.

The following observations support this view:

1. Most of the patients survived six to twelve hours after being wounded, during which time they had not coughed effectively.
2. Active bleeding was not found at bronchoscopy in any instance.
3. The relief that followed aspiration usually lasted several hours.
4. Instrumentation was never observed to increase hemorrhage.

CLINICAL FEATURES

Serious intrabronchial hemorrhage does not present a diagnostic problem. In severe instances there were audible tracheal râles, orthopnea, and cyanosis. In addition, disorientation, excitement, and restlessness, the results of cerebral anoxia, were commonly present. The physical and x-ray examination revealed signs of the hemothorax or hemopneumothorax which was invariably present. Subcutaneous emphysema was frequent. More important was the presence of palpable râles. The latter is a sign of great value. In penetrating chest wounds this may rarely be due to pulmonary edema but for practical purposes it was diagnostic of retained bloody secretions in the tracheobronchial tree, causing serious bronchial obstruction which can be most effectively relieved by bronchoscopy. It also offers a simple and rapid method of determining the affected lung, and roughly delineating the lobes involved. As previously noted, the most severe anoxia was seen in those in whom there had been a "spill" of bloody secretions into the unaffected lung.

TREATMENT

Bronchial hemorrhage producing anoxia of the gravity described requires prompt treatment. Clearing the tracheobronchial tree takes precedence over all other resuscitative and diagnostic measures. Aspiration of the secretions should be done regardless of the patient's condition. Suction through an intratracheal catheter is valuable to accomplish this objective. Greatly to be preferred, however, is bronchoscopic aspiration which permits visualization of the branch bronchi of the individual lobes. Experience reported here permits this assertion to be made categorically. Secretions producing "palpable râles" over a bronchopulmonary segment, which could not be aspirated through an intratracheal catheter, have been repeatedly removed through the bronchoscope.

The patient's condition may be precarious when he is first seen. His head and shoulders have usually already been elevated, if such is the case, and oxygen is being administered. It is not safe to lower the head of such patients or even temporarily stop the administration of oxygen. These men have been bronchoscoped on army cots in approximately the position in which they lay. It is possible to visualize the main bronchi readily in this position by having an assistant lift the chest slightly (Fig. 1). Oxygen administration is started by the nasal catheter method before passage of the instrument and preparation is made to change the flow to the sidearm of the bronchoscope immediately after passing the glottis. Failure to observe this precaution may result in a fatality during instrumentation. Aspiration usually yields 30 to 60 c.c. of blood and mucus secretion. The chest should be palpated again after clearing the trachea and the main bronchi. "Palpable râles" frequently remain over one or more lobes depite aspiration of large amounts of secretion. It is then advantageous to apply the aspirator tip directly to the orifice of the affected lobe. This excites cough and usually the secretions are expelled and immediately withdrawn. No râles can be felt at the completion of a properly conducted bronchoscopy.

Subsequent management required definitive treatment for the associated injuries. In the exceptional patient there was only a minor wound of the chest wall and lung associated with a hemothorax. Debridement of the former and aspiration of the latter may be the only treatment required in injuries of this type. More commonly, there were associated sucking wounds, lacerations of the lungs, tears of the diaphragm, or a thoracoabdominal wound. Tension pneumothorax was only occasionally observed. The above wounds required appropriate surgical treatment which will not be discussed here. It was found that it was usually preferable to operate soon after clearing the air passages, provided a satisfactory response was made and shock was not present.

It was necessary occasionally to do another bronchoscopic aspiration after anesthesia had been induced before introducing an intratracheal catheter. Bronchoscopic aspiration at the end of operations was done routinely. These patients, if encouraged and assisted, were usually able to cough after operation. Occasionally this was not the case and in these, prompt bronchoscopic aspiration was repeated as often as necessary until the bleeding stopped or effective cough was re-established.

TECHNIQUE

Battle conditions require improvisation. The first endoscopies were done without trained assistants or anesthesia. As already noted, many patients were on army cots, oxygen was being administered, and the patient's condition was of such gravity that he could not be moved. It was found that these patients should be "bronchoscoped where they lay." Under these circumstances it was learned that satisfactory endoscopy could be done in unconventional positions and that anesthesia was unnecessary. It is now felt that for the following reasons, anesthesia is not only unnecessary, but actually undesirable:

1. Valuable time is consumed which might otherwise be used in operating upon other gravely wounded patients.
2. Efforts to apply an anesthetic may produce ineffectual cough. This exposes the patient to the danger of displacement of secretions into the remaining functioning lung with disastrous results.
3. Active cough reflex during instrumentation preserves the "tussive squeeze"[16] and greatly assists in emptying the small bronchi.
4. Even prolonged efforts to produce anesthesia are comparatively ineffectual because of excessive amounts of secretion in the airways.
5. No instance of failure to pass the bronchoscope, or injury to the patient was experienced, despite occasional complete lack of cooperation in robust and disoriented patients.

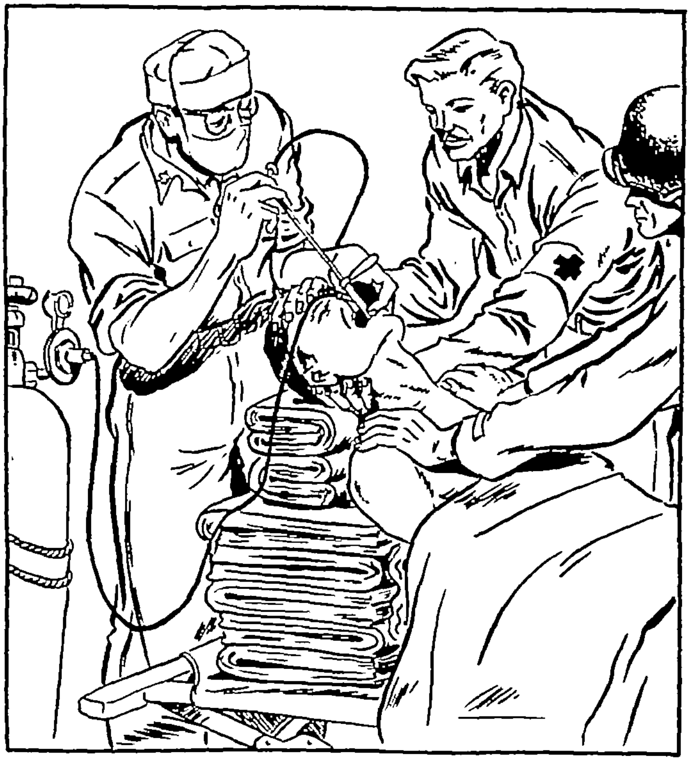

Fig. 1.—Position used for bronchoscopic aspiration in seriously anoxic patients. Note that oxygen is being administered through the bronchoscope.

Reference has already been made to an unconventional position used in bronchoscoping patients on a cot. More commonly, aspiration was done on an improvised operating table made with a litter. Here it was advantageous merely to elevate the patient's head in introducing the instrument (Fig. 2). Visualization of the lower bronchi necessitated raising the patient's chest slightly since the head could not be lowered. Flagg's laryngoscope, which eliminates one wire connection, was found preferable.

MORTALITY

Indications among combat casualties for active measures to remove blood and secretions from the tracheobronchial tree arose invariably in patients presenting lacerations of the lung and sucking wounds. Three of the seven operative deaths that occurred in the group were directly or indirectly attributable to intrabronchial bleeding. One patient who had a massive hemorrhage into the bronchi during operation will be described (Case 4). The others had severe and persistent hemoptysis after the operation. Cough was fairly effectual but postoperative pneumonia developed and death occurred on the seventh and eighth postoperative day. A fourth patient died suddenly twenty-four hours after operation, presumably from tension pneumothorax. He was operated upon during a period before closed intercostal subaqueous drainage was routinely established at operation; of the remaining three, one died of shock during the operation, a second whose shock was never corrected before

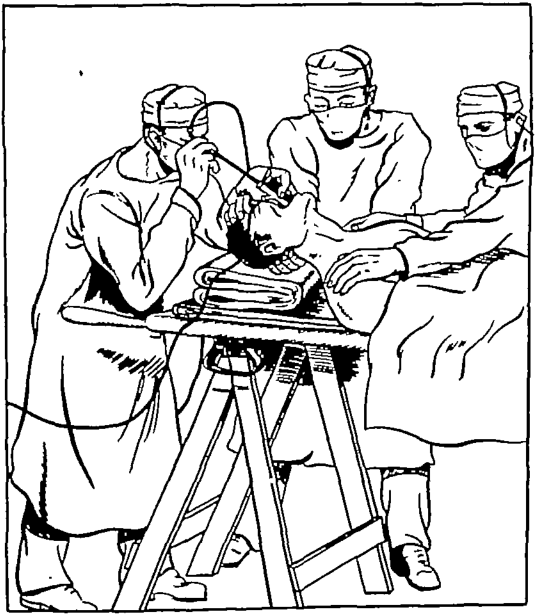

Fig. 2.—Position of patient after bronchoscope is in place for aspiration of lower bronchi on an improvised operating table.

2. The experience recorded indicates that cases of this type occur frequently in hospitals operating near the front when proper first aid is applied and the casualties are promptly evacuated.

3. It has been shown that in some patients aspiration of the tracheobronchial tree is practically the only definitive treatment necessary. It constitutes an essential part of the resuscitative treatment and anesthesia in those who are more severely wounded.

4. Two techniques of bronchoscopic aspiration applicable to surgery in the field have been described; the advantages of instrumentation without anesthesia have been considered. It is emphasized that the gravity of the patient's condition does not contraindicate such treatment. Indeed, the more grave his condition, the more urgent is the necessity for such aspiration.

5. Bronchoscopic aspiration has never been observed to increase intrabronchial bleeding and repeated endoscopies are tolerated without ill effect.

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THORACOSCOPY AND PNEUMONOLYSIS IN PULMONARY TUBERCULOSIS

NOTES ON TECHNIQUE AND IMMEDIATE RESULTS

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THE following study is based upon the experience gained in 500 cases, with 654 operations performed, during the period of 1934 to 1945 by several thoracic surgeons at the Grace Dart Home Hospital, the Royal Edward Laurentian Hospital, and three general hospitals in Montreal. It includes operations on Army and ex-Army personnel carried out by the author in 1943 and 1944 under arrangement with the Department of Veterans Affairs. An additional number (100 operations in seventy-six cases) was performed since the preparation of this paper and is not considered in the postoperative statistics.

It is not intended to give a complete analysis of the cases with a follow-up study of the clinical results. This task has to be postponed until a sufficient period of time has elapsed to allow for an accurate account of the end results. Many details about instruments, technique, etc. have to be omitted because of lack of space. It is the writer's intention, however, to take a definite stand regarding the major problems in technique and with reference to the important and difficult task of choosing the right cases and the right adhesions for this operation.

INSTRUMENTS

During the first five years of our series of operations we used the instruments designed by Jacobaeus, with the modifications of Unverricht. In later years we changed over to the Coryllos thoracoscope. When high frequency electrodes were advisable we used the unit made by the same manufacturer (A.C.M.I.). In addition to these instruments, a cautery blade bent into the shape of a hook and a suction tube are occasionally very useful.

ADHESIONS

Table I shows the different types of adhesions that were encountered in our cases. Adhesions containing blood vessels were not considered separately. Blood vessels prevented a complete separation in only a few earlier cases (0.8 per cent). The type of adhesion is brought into relation with the operative procedure and it is clearly shown how the success of pneumonolysis diminishes with the more difficult type of adhesions. It is necessary to define the terms used in the table. *Complete pneumonolysis* means that all visible adhesions were severed. *Partial pneumonolysis* means that either some adhesions were

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cut completely and others left alone on account of difficulties which were encountered, or that an extensive adhesion, usually a web adhesion in the posterior mediastinum or a spool adhesion at the apex, were cut partially to the extreme point of apparent safety. *Thoracoscopy* means that the visualized adhesion was not suitable for cutting or that no adhesions were found.

Solitary or multiple string and band adhesions were usually found between lateral, anterior, or posterior chest wall and the convexity of the lobes (upper lobe most frequently). We encountered spool adhesions chiefly between the apex of the upper lobe and the pleural cupola (first rib and subclavian vessels). Web adhesions and shelves were more numerous posteriorly in the mediastinal gutter. Adhesions to the diaphragm were usually string- or bandlike in character and ran from the outer lower border of the lung to the costophrenic angle. Diffuse adhesions from the base of the lung to the diaphragm were presumed by their x-ray appearance and when the lung could not be lifted by means of a blunt ribbon retractor.

TECHNIQUE OF PNEUMONOLYSIS

Anesthesia.—All patients received ¼ grain morphine and 1/100 grain scopolamine one-half hour before operation. We prefer local anesthesia. A general anesthetic (N_2O) was used only in a very few cases and did not give encouraging results.

Introduction of Instruments.—Most adhesions are situated in the posterior or axillary region. The lung, therefore, collapses in a rather anteroposterior direction and allows sufficient air space in the anterior chest. Posteroanterior x-rays may show only a very small zone of pneumothorax, while oblique x-rays, tomographic or stereoscopic views, may indicate better the most suitable place for the introduction of the trocar.

We introduce the scope according to the individual case. In 488 instances out of 754 stages the first puncture was made in the anterior axillary line at the fifth interspace. In the remainder, the trocar was inserted anywhere between midclavicular and midscapular line.

The inserted scope indicates where the cannula for the cautery should be introduced. In the great majority of operations, the fifth to the seventh interspace in the posterior axillary line was chosen.

In the posterior half of the chest, the intercostal vessels and nerves are protected by the lower rib margin. If, therefore, the trocar be inserted in an oblique direction from above downward, there is little likelihood of damage. Anterior vessels and nerves are exposed, but they can be easily pushed away as the spaces between the ribs are widened.

Almost all postoperative hemorrhages into the chest cavity are due to bleeding from the trocar opening (intercostal vessels) and rarely from a severed adhesion. The wall of the trocar exercises a temporary hemostasis while withdrawal of the instrument invites new bleeding. We have observed this event on several occasions through the thoracoscope.

TABLE I. TYPE OF ADHESION IN RELATION TO OPERATIVE PROCEDURE IN 576 OPERATIONS (TWENTY-ONE BILATERAL)

	OPERATIVE PROCEDURE									TOTAL	
	COMPLETE PNEUMONOLYSIS			PARTIAL PNEUMONOLYSIS			THORACOSCOPY				
TYPE OF ADHESION	CASES	% ADHESIONS	% PROCEDURE	CASES	% ADHESIONS	% PROCEDURE	CASES	% ADHESIONS	% PROCEDURE	CASES	PER CENT
String, band, and cord adhesions	141	60.0	88.7	17	5.6	10.7	1	1.8	0.6	159	26.6
Same, combined with web and shelf adhesions (not mediastinal)	66	28.1	42.0	86	28.1	54.8	5	8.9	3.2	157	26.3
Solitary or combined adhesions, the majority of which are:											
Spool adhesions	18	7.7	15.8	76	24.8	66.7	20	35.7	17.5	114	19.1
Mediastinal adhesions	2	0.8	2.2	79	25.8	87.8	9	16.2	10.0	90	15.1
Diaphragmatic adhesions	0	0.0	0.0	13	4.3	100.0	0	0.0	0.0	13	2.2
Diffuse adhesions or partial synechia of lung to chest wall	8	3.4	13.6	35	11.4	59.2	16	28.5	27.2	59	9.9
No adhesions found	0	0.0	0.0	0	0.0	0.0	5	8.9	100.0	5	0.8
Total	235	39.4		306	51.2		56	9.4		597	100.0

Evaluation of Adhesions.—After aspiration of the fluid, if such be present, inspection of the adhesions is necessary. Their position should be carefully checked with the last available x-ray. An adhesion still visible in the x-ray may have ruptured before the operation (5 cases). Very often, adhesions to healthy lobes are rather useful as they allow a selective collapse of the diseased parts after division of the adhesions to the affected lobes. Selective collapse is especially important in those cases where any collapse therapy on the contralateral side is contemplated or already performed. The presence of a peripheral cavity in the x-ray demands special precautions. The possibility of a tension cavity should be kept in mind, since separation of the adhesion may be followed by rupture of the cavity into the pleural space.

Separation of Adhesions.—Adhesions which may contain lung tissue are usually divided by decollement of the parietal pleura. Occasionally this procedure may be assisted by the hydraulic pressure of injected fluid (novocaine or saline) into the plane of cleavage. For thick adhesions which cannot be transilluminated, or mediastinal adhesions, the hook cautery is very useful. It picks up small layers and cuts them separately. In small string adhesions, distant from the optic and dangerously near to the great vessels, "hooking" serves the same purpose.

Special attention should be given to blood vessels within the adhesion. Within the last 10 years, the galvanocautery became a stepchild while the high frequency cautery (Matson) gained more and more popularity. A word in favor of the galvanocautery may be said.

A cautery blade, red hot and used at its edge, will deposit a great amount of heat at one very small spot. The tissue, especially in a string or band adhesion, will be charred in the immediate neighborhood, but the dried parts will prevent the further conduction of heat to remote parts of the adhesion. If there is a blood vessel within the adhesion, it will be severed, but the very narrow zone of charred tissue will rather prevent a contraction of the vessel wall and the small clot is not firm enough to stop the blood flow. Bleeding can be expected even hours after the operation. This method, therefore, is not recommended.

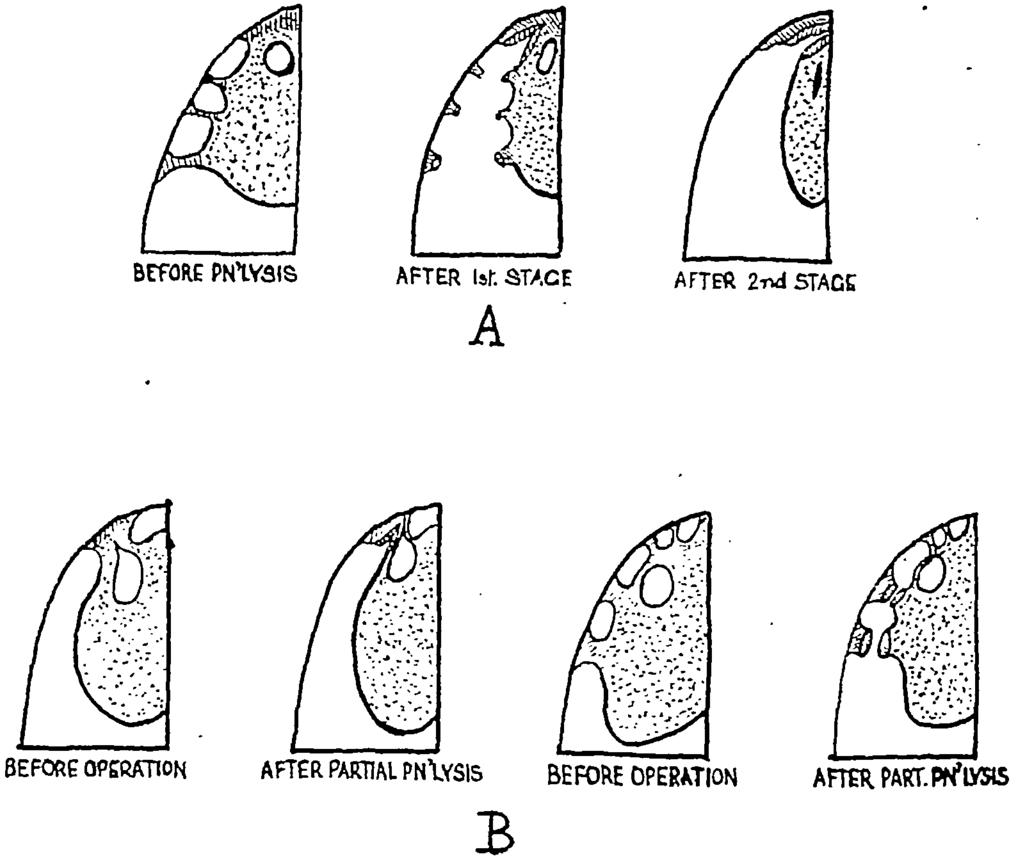

Fig. 1.—Partial pneumonolysis. *A*, pneumothorax should be continued and a further stage attempted. *B*, pneumothorax should be abandoned immediately.

The application of the cautery with its flat side to the adhesion and dull red (visible only in the dark) will heat up the adhesion slowly and uniformly. It will cause coagulation of a larger field, even if the adhesion contains a blood vessel of considerable size. The edge of the cautery will readily cut through the coagulated zone while the blood vessels are well secured by coagulation and subsequent shrinking of the surrounding tissue. The high frequency cautery has its place at special occasions but it is by no means the key to a successful separation of blood vessels.

In many instances, adhesions can be severed only partially, due to their shortness or the lack of a free edge to work on, or they are so diffuse that the operation extends over several hours and the patient becomes uncomfortable. The question arises whether to abandon any further attempt or to try separating again at a later stage. No fast rules can be given. If the remaining adhesion is near a peripheral cavity, it is far better to abandon the pneumothorax *immediately (Fig. 1, A and B). If there is no cavity in the neighborhood of* the adhesion and the visceral pleura appears healthy, the pneumothorax could be continued and a further stage of pneumonolysis attempted.

Closure of Trocar Opening.—Intrapleural pressures should be adjusted before or immediately after closure of the trocar openings and should be on the negative side. The closure of the wound itself is usually carried out by one or two sutures or clips. This type of closure, however, is far from ideal as it does not include the parietal pleura. No air will escape or enter between the outside and the thoracic cavity, but air may enter into the fascial planes from the inside and cause a subcutaneous emphysema. Its extent depends on the intrapleural pressures and the size and number of trocar openings. None of the methods of closure described in the literature have been successful in the prevention of subcutaneous emphysema in our experience. Recently, we tried the high frequency current for coagulation of the wall of the trocar opening as described by Vineberg and Kunstler for the intracavitary suction drainage. We believe that this method may reduce the incidence of emphysema and hemorrhage after withdrawal of the instruments.

Postoperative Care.—Special attention is paid to the fever chart. Failure of fever to subside after a week or a rise of temperature forty-eight hours after the operation is considered with suspicion. Routinely inspiratory and expiratory x-rays are made the next day in spite of the sometimes difficult interpretation due to subcutaneous emphysema. Refills are accomplished depending upon the information thus obtained.

AGE AND SEX

In 576 cases we had 282 male and 294 *female patients. Our oldest patient* was 54 years, the youngest a child of 3 years. Most of the male patients were between 21 and 25 years old, most of our female patients between 15 and 30 years old (Fig. 2). Relation of sex and side of operation is shown in Table II.

TABLE II. RELATION OF SEX TO SIDE OF OPERATION (576 CASES)

SEX	LEFT	RIGHT	BILATERAL	TOTAL NO.	TOTAL %
Male	130	144	8	282	49
Female	155	126	13	294	51
Total	285	270	21	576	100

STAGE OF DISEASE

Of 576 cases, twenty-one had bilateral pneumothorax and bilateral pneumonolysis. Of the remaining 555, a fair number of cases with bilateral disease had pneumothorax on the other side without thoracoscopy or pneumonolysis. Others with bilateral disease had interruption of the phrenic nerve, transthoracic suction drainage, or thoracoplasty on the opposite side. Table III shows the stage of disease in detail.

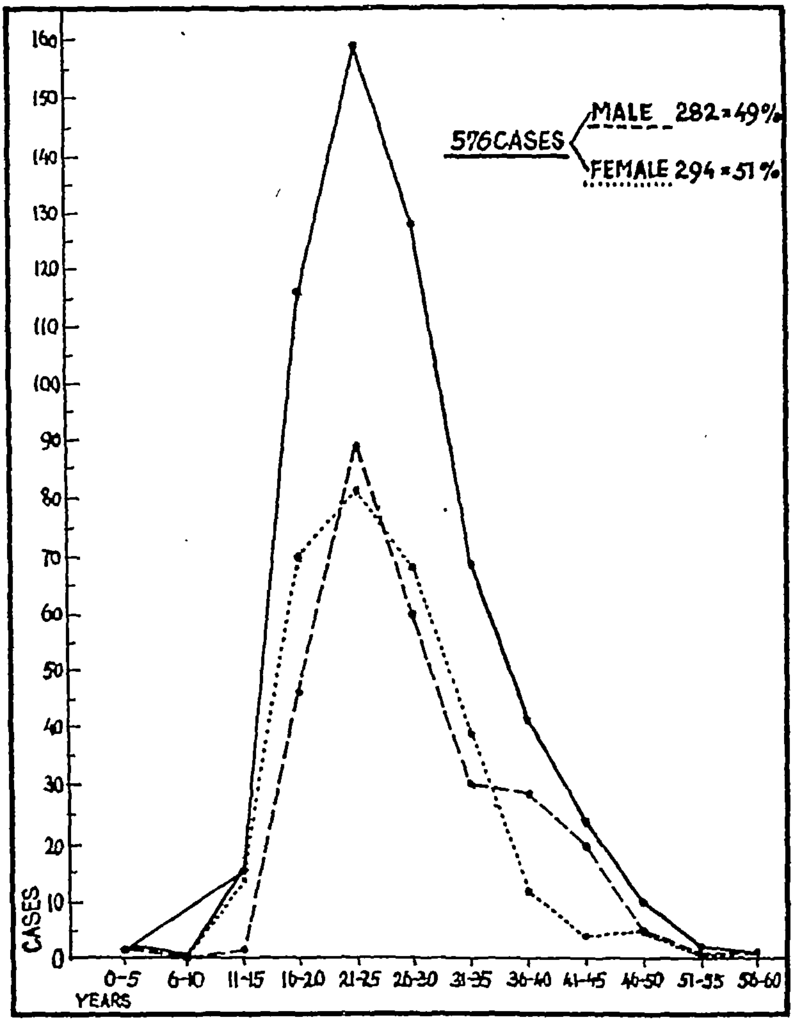

Fig. 2.—Distribution of patients according to sex and age.

Fig. 3 reveals that in the course of the years unilateral pneumonolysis was attempted in increasing number for moderately and far-advanced cases.

IMMEDIATE RESULTS

It is not easy to determine the result of a pneumonolysis, because complications may develop independently. The clinical result depends also on the

TABLE III. STAGE AND DISTRIBUTION OF DISEASE IN 576 PATIENTS WITH THORACOSCOPY AND PNEUMONOLYSIS

	MILD	MODERATELY ADVANCED	FAR ADVANCED	TOTAL
Unilateral thoracoscopy and pneumonolysis (555 cases)				
Unilateral disease	44	208	61	313
Bilateral disease	--	80	162	242
Bilateral thoracoscopy and pneumonolysis	--	4	17	21
Total	44 (7.6%)	292 (50.7%)	240 (41.7%)	576 (100%)

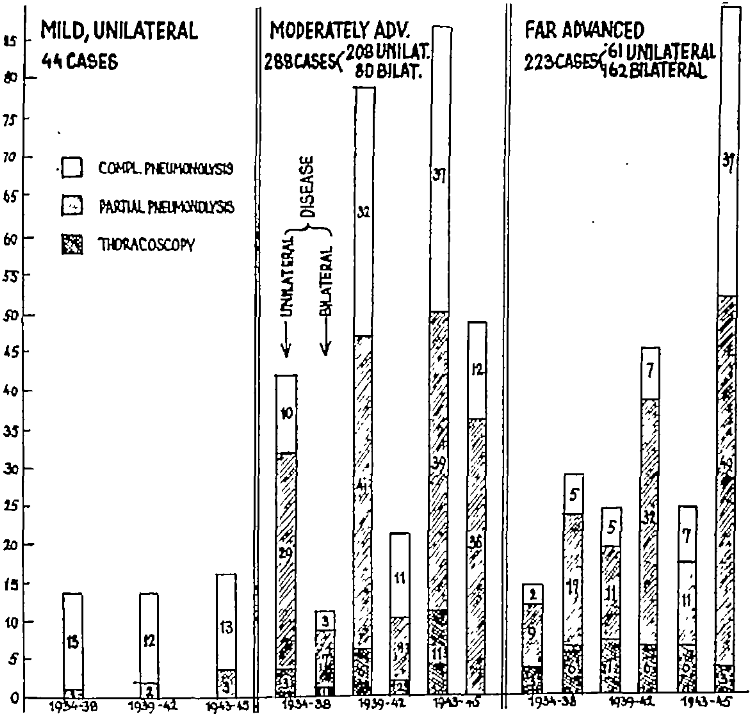

Fig. 3.—555 patients with unilateral thoracoscopy and pneumonolysis from 1934 to 1945.

condition of the other lung, so that only cases with the controlateral side free from disease or under control are able to give us a true picture of the result achieved. Pneumonolysis is not a therapeutic measure per se, but only an adjuvant to improving an inefficient pneumothorax. The simplest method of classification is to connect the immediate results with the effect of the pneu-

TABLE IV. IMMEDIATE RESULTS OF UNILATERAL THORACOSCOPY AND PNEUMONOLYSIS (479 CASES, 602 STAGES): RELATION TO NUMBER OF STAGES, TYPE OF OPERATION, FATE OF PNEUMOTHORAX A ND COMPLICATIONS WITHIN SIX MONTHS AFTER OPERATION

					FATE OF PNEUMOTHORAX FOR SIX MONTHS AFTER OPERATION										
		OPERATION				UNSATISFACTORY CASES									
							COMPLICATIONS								
STAGES	CASES	COMPLETE PNEUMONOLYSIS	PARTIAL PNEUMONOLYSIS	THORACOSCOPY	SATISFACTORY CASES	TOTAL	DANGEROUS NONCUTTABLE ADHESIONS	TENSION CAVITY	ADHESIVE PLEURITIS	FLUID	LEFT AGAINST ADVICE, REFUSED TREATMENT	SPONTANEOUS PNEUMOTHORAX BRONCHOPLEURAL FISTULA		EMPYEMA	DEATHS
I	385 (80.3%)	152	187	46	287 (74.5%)	98 (25.5%)	43	7	12	7	11	3	6	9	6
II	70 (14.6%)	19	47	4	50 (71.4%)	20 (28.6%)	7	1	3	2	1	0	1	4	2*
III	19 (4.0%)	3	16	0	13 (68.4%)	6 (31.6%)	4	0	1	0	0	0	0	1	0
IV	5 (1.1%)	3	2	0	4 (80.0%)	1 (20.0%)	0	0	1	0	0	0	0	0	0
Total	479 (100%)	177 (37.1%)	252 (52.5%)	50 (10.4%)	354 (73.8%)	125 (26.2%)	54	8	17	9	12	3	7	14	8

*One patient died after pneumonectomy performed four months after the second stage of partial pneumonolysis.

TABLE V. IMMEDIATE RESULTS OF BILATERAL THORACOSCOPY AND PNEUMONOLYSIS (21 CASES, 52 STAGES): RELATION TO NUMBER OF STAGES, TYPE OF OPERATION, FATE OF PNEUMOTHORAX AND COMPLICATIONS WITHIN SIX MONTHS AFTER OPERATION

STAGES	SIDES	OPERATION			FATE OF PNEUMOTHORAX FOR SIX MONTHS AFTER OPERATION			
						UNSATISFACTORY (SIDES)		
		COMPLETE PNEUMONOLYSIS	PARTIAL PNEUMONOLYSIS	THORACOSCOPY	SATISFACTORY (SIDES)	TOTAL	FLUID	ADHESIONS UNCUTTABLE
I	32 (76%)	25 (78.1%)	5 (15.6%)	2 (6.3%)	29 (90.6%)	3 (9.4%)	0	3
II	10 (24%)	4 (40%)	6 (60%)	0 –	9 (90%)	1 (10%)	1	0
Total	42 (100%)	29 (69%)	11 (26.2%)	2 (4.8%)	38 (90.5%)	4 (9.5%)	1	3

monolysis on the pneumothorax. A period of six months after operation seems to be sufficient to see the effect of the pneumothorax (closure of cavity, conversion of sputum, etc.). We may therefore stipulate the results in the following manner: thoracoscopy or pneumonolysis is satisfactory if a pneumothorax can be safely continued for a duration of six months; if a pneumothorax has to be abandoned within six months, the verdict should be unsatisfactory.

There were 500 patients with 521 sides in our series, observed for more than six months. 392 cases were satisfactory and 120 unsatisfactory, the ratio being 75.2 per cent and 24.8 per cent. Fig. 4 shows these figures in relation to the type of operation.

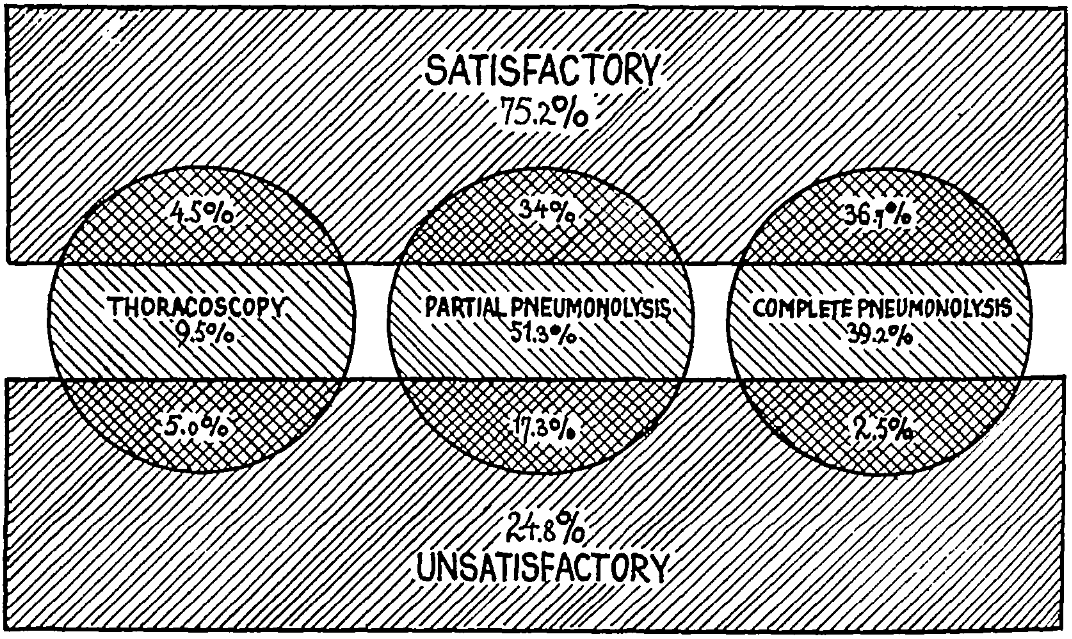

Fig. 4.—Results of complete or partial pneumonolysis and thoracoscopy in 500 patients (521 sides) six months after operation.

If we list the twenty-one cases with bilateral pneumonolysis separately, we arrive at the following figures: there were 90.5 per cent satisfactory and 9.5 per cent unsatisfactory results; the high percentage of success in these cases may be attributed to surgical daring in the face of far-advanced bilateral disease.

Of 479 cases of unilateral thoracoscopy or pneumonolysis, 385 had a one-stage operation, while the balance needed two or more stages. As Table IV illustrates, the satisfactory results diminish very little with the number of stages.

Of the twenty-one cases of bilateral operations, thirty-two sides had a one-stage operation and ten required two stages. There were no deaths nor major complications within six months in this group (Table V).

COMPLICATIONS

Complications may be due directly to the operation or may arise independently. It is very difficult to set a fast line. A spontaneous pneumothorax may develop two weeks after operation but the fistula may be in a place different from that of the severed adhesion (Fig. 5). We may classify all complications arising within four weeks after operation as postoperative, while for any complication after that interval the operation should not be blamed.

The main complications are the following: (1) considerable subcutaneous emphysema; (2) accumulation of fluid in the chest cavity; (3) hemorrhage; (4) spontaneous pneumothorax with or without persistent bronchopleural fistula; (5) empyema; and (6) adhesive pleuritis.

Subcutaneous Emphysema (See Table VI).—No treatment was given in the first and second degree. In the third degree, it was necessary to treat the underlying cause, which was usually increased intrathoracic pressure or tension pneumothorax (removal of air, administration of oxygen). Fig. 6 shows a case where the emphysema affected the whole chest wall, neck, and face. The eyelids could not be opened until the fifth day after operation.

TABLE VI. INCIDENCE OF SUBCUTANEOUS EMPHYSEMA AFTER THORACOSCOPY AND PNEUMONOLYSIS

DEGREE	CASES	PER CENT
Slight	103	25.6
Moderate	34	8.5
Severe	7	1.8
None	256	64.1

Fluid Accumulation.—In 500 patients (521 sides), we encountered fluid in 291 instances. Tables VII and VIII show in detail the development and/or modification of fluid in relation to the postoperative period and the type of operation. In the multistage operations, only the fluid before the first stage and the condition after the last stage have been considered. There were 240 patients without fluid in the pleural space before operation, while four weeks to six months after the operation the number of patients without fluid increased to 374, or 73 per cent of all patients. Most of the patients with fluid for six months or longer after the operation had only partial pneumonolysis, or thoracoscopy showed adhesions unsuitable for cutting. Multiple stages more frequently caused fluid than single stages. If a multiple stage operation resulted in a complete pneumonolysis, the incidence of fluid almost reduced to that of single-stage operations.

Pleural effusions, especially larger ones and those following a hemorrhage, are usually accompanied by elevation of temperature. We noticed that there is a frequent relationship between the nature of the fluid and the height and duration of fever. Unless the fluid is hemorrhagic and due to operation, all other patients with high and long-standing fever will eventually develop em-

TABLE VII. DEVELOPMENT AND/OR MODIFICATION OF FLUID AFTER THORACOSCOPY AND PNEUMONOLYSIS: SINGLE STAGE OPERATION, 417 CASES

OPERATION	FLUID BEFORE OPERATION		FLUID WITHIN 4 WEEKS AFTER OPERATION					FROM 4 WEEKS TO 6 MONTHS AFTER OPERATION					
	AMOUNT	CASES	NONE	MINIMAL	MODERATE	CLOUDY	PUS	NONE	MINIMAL	MODERATE	LARGE	CLOUDY	PUS
Complete Pneumo-.nolysis	None	99	69	24	6	0	0	91	5	2	0	1	0
	Minimal	74	12	49	12	1	0	49	18	2	2	0	3
	Moderate	4	0	3	1	0	0	4	0	0	0	0	0
	Total	177 (42.5%)	81 (45.7%)	76 (43%)	19 (10.7%)	1 (0.6%)	0 (0%)	144 (81.3%)	23 (13%)	4 (2.3%)	2 (1.1%)	1 (0.6%)	3 (1.7%)
Partial Pneumo-nolysis	None	78	61	9	8	0	0	71	5	0	0	1	1
	Minimal	92*	8	58	26	0	0	60	22	2	2	1	2
	Moderate	18	0	8	10	0	0	5	8	2	1	0	2
	Large	3	0	0	3	0	0	0	0	1	1	1	0
	Pus	1	0	0	0	0	1	0	0	0	0	0	1
	Total	192 (46%)	69 (35.9%)	75 (39.1%)	47 (24.5%)	0 (0%)	1 (0.5%)	136 (70.9%)	35 (18.2%)	5 (2.6%)	4 (2.1%)	3 (1.5%)	6 (3.1%)
Thoracos-copy	None	23	21	2	0	0	0	21	2	0	0	0	0
	Minimal	17	2	11	4	0	0	9	5	0	1	1	1
	Moderate	5	0	3	2	0	0	0	0	1	4	0	0
	Large	2	0	0	2	0	0	1	0	1	0	0	0
	Pus	1	0	0	0	0	1	0	0	0	0	0	1
	Total	48 (11.5%)	23 (47.9%)	16 (33.3%)	8 (16.7%)	0 (0%)	1 (2.1%)	31 (64.5%)	7 (14.6%)	2 (4.2%)	5 (10.4%)	1 (2.1%)	2 (4.2%)
Total		417 (100%)	173 (41.5%)	167 (40.1%)	74 (17.7%)	1 (0.2%)	2 (0.5%)	311 (74.7%)	65 (15.6%)	11 (2.6%)	11 2.6%)	5 (1.2%)	11 (2.6%)

*Three patients died within four weeks after operation. (0.7 per cent.)

TABLE VIII. DEVELOPMENT AND/OR MODIFICATION OF FLUID AFTER THORACOSCOPY AND PNEUMONOLYSIS: MULTIPLE STAGE OPERATION, 104 CASES*

OPERATION	FLUID BEFORE OPERATION		FLUID WITHIN 4 WEEKS AFTER OPERATION				FROM 4 WEEKS TO 6 MONTHS AFTER OPERATION				
	AMOUNT	CASES	NONE	MINIMAL	MODERATE	LARGE	NONE	MINIMAL	MODERATE	CLOUDY	PUS
Complete Pneumonolysis	*None*	13	*11*	2	0	0	13	0	0	0	0
	Minimal	8	3	5	0	0	7	1	0	0	0
	Moderate	8	1	1	6	0	2	6	0	0	0
	Total	29 (27.9%)	15 (51.7%)	8 (27.6%)	6 (20.7%)	0 (0%)	22 (75.8%)	7 (24.2%)	0 (0%)	0 (0%)	0 (0%)
Partial Pneumonolysis	None	27	11	5	11	0	15	6	3	1	2
	Minimal	27	7	10	8	2	16	8	2	0	1
	Moderate	14	2	9	2	1	8	5	0	1	0
	Large	3	1	1	0	1	1	1	0	1	0
	Total	71 (68.2%)	21 (29.6%)	25 (35.2%)	21 (29.6%)	4 (5.6%)	40 (56.4%)	20 (28.2%)	5 (7%)	3 (4.2%)	3 (4.2%)
Thoracoscopy	Minimal	4	0	4	0	0	1	2	1	0	0
	Total	4 (3.9%)	0 (0%)	4 (100%)	0 (0%)	0 (0%)	1 (25%)	2 (50%)	1 (25%)	0 (0%)	0 (0%)
Total		104 (100%)	36 (34.6%)	37 (35.6%)	27 (25.9%)	4 (3.9%)	63 (60.5%)	29 (27.9%)	6 (5.8%)	3 (2.9%)	3 (2.9%)

*In order not to complicate the table, only the state of fluid before the first and after the last stage is considered.

pyema. If the onset of temperature is sudden, we have to suspect a traumatic lesion of the lung parenchyma (minute bronchopleural fistula), even if the fluid is clear for weeks and without demonstrable microorganisms. If such a pneumothorax is not abandoned at once, the change of the fluid into pus is the rule. Fluid with low-grade fever usually disappears if the underlying cause (adhesions, blood) is removed. Pleural effusion deriving from infection of the pleura does not show a sudden rise in temperature but causes a rather slow and steady elevation.

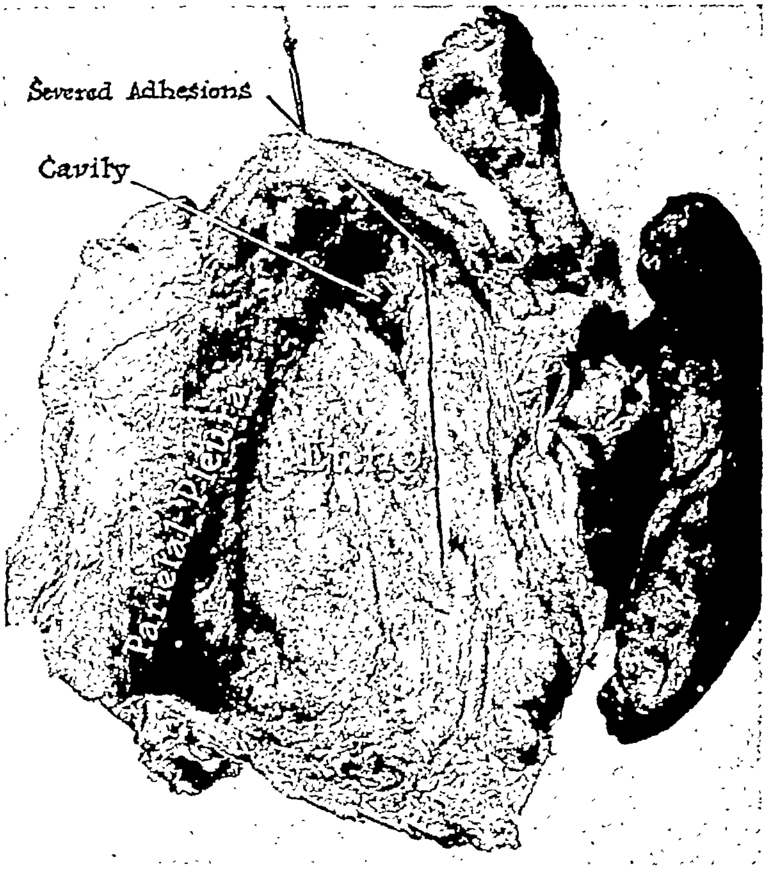

Fig. 5.—Rupture of cavity with spontaneous pneumothorax and fatal hemorrhage: cavity distant to severed adhesion.

Hemorrhage.—The incidence of hemorrhage in 754 operations was small and remained under the average reported by other authors. Table IX shows the number of hemorrhages.

There were no fatalities due to hemorrhage, and the loss of blood amounted to more than 100 c.c. in only eleven patients. In one patient the postoperative hemorrhage from a severed adhesion was so severe that the pleural cavity had to be packed. In another patient, a terminal hemorrhage occurred due to rupture of a cavity into the pleural space with no connection with a partial pneumonolysis two weeks before.

Prevention and treatment of hemorrhage during operation have already been discussed. Treatment of postoperative hemorrhage consisted in frequent and complete aspirations and partial replacement of fluid by air. Instillation of penicillin was carried out.

The incidence of hemorrhage falls as the experience of the operator increases.

Spontaneous Pneumothorax and Bronchopleural Fistula.—Out of fifteen cases of spontaneous pneumothorax, six were due to ruptured adhesions which contained lung tissue (in two instances the rupture occurred before operation). Pneumonolysis was directly to be blamed in one case, leaving the residual adhesion under strain and with little support. Increasing of intrapleural pressure in order to "stretch" the adhesion aided greatly in the development of this complication.

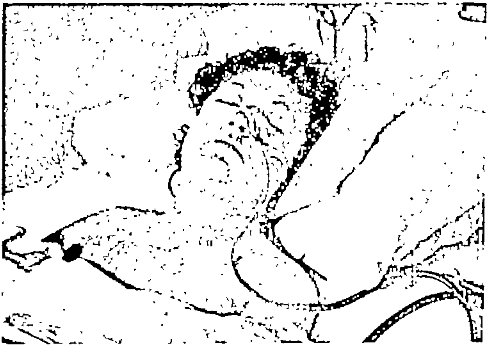

Fig. 6.—Severe degree of subcutaneous emphysema after partial pneumonolysis.

Rupture of a tuberculous cavity was to be blamed in six instances. Three of them occurred in the four-week period after operation, two five weeks after, and one ten days before thoracoscopy. One patient had a complete pneumonolysis and in another at autopsy showed rupture of a cavity away from the partially severed adhesions. In both cases we probably dealt with tension cavities.

TABLE IX. INCIDENCE OF HEMORRHAGE IN THORACOSCOPY AND PNEUMONOLYSIS

HEMORRHAGE	CASES	SLIGHT	MODERATE	SEVERE
During operation				
Trocar puncture	3	3	0	0
Severed adhesions	10	6	4	0
Total	13	9	4	0
Postoperative	12	5	4	3*
Total	25	14	8	3
Per cent to all operations (754)	3.3	1.8	1.1	0.4

*One patient with a severe postoperative hemorrhage from a severed adhesion.

TABLE X. SPONTANEOUS PNEUMOTHORAX WITH OR WITHOUT BRONCHOPLEURAL FISTULA (15 CASES)

CASE	EXTENSION OF DISEASE	OPERATION	ONSET OF SPONTANEOUS PNEUMOTHORAX	PERSISTENT BRONCHOPLEURAL FISTULA	REASON	PX CONT. FOR 6 MONTHS	PN'X DISCONTINUED FOR		DIED WITHIN 6 MONTHS	REMARKS
							FLUID	EMPYEMA		
22	unl fa	PPl	5 months ao	no	r res adh	yes	no	no	no	—
26	bil ma	PPl	2 months ao	yes	r res adh	no	no	tuberculous	no	Pressures increased after PPl.
48	bil fa	PPl	5 days ao (2nd st)	no	inj to l. by caut.	yes	no	no	no	Had two more stages: CPl.
71	bil fa	PPl	6 weeks ao	yes	r res adh	no	no	mixed	no	—
74	bil fa	PPl	3 days ao	yes	r cavity	no	no	tuberculous	no	Pressures increased after PPl.
78	bil fa	CPl	6 days ao	yes	r cavity	no	no	mixed	no	Tension cavity; refused thoracoplasty
98	bil fa	CPl	6 hours ao	no	inj to l. by caut.	yes	no	no	no	—
196	bil fa	PPl	10 days bo	yes	r cavity	no	no	mixed	no	Bronchopleural fistula closed by endoscopic operation (Fig. 15)
330	bil ma	PPl	3 months bo	no	r of adh	yes	no	no	no	Failure of having adhesion cut in time
247	bil fa	PPl	2 weeks ao	yes	r res adh	no	no	no	yes	4 days after onset of spontaneous pneumothorax
324	unl ma	PPl	10 hours ao	no	inj to l. by caut.	no	yes	no	no	—
358	unl ma	Th	2 months bo	yes	r of adh.	no	no	tuberculous	no	Failure of having adhesion cut in time. Thoracoplasty
420	unl ma	PPl	5 weeks ao	yes	r cavity	no	no	mixed	no	—
442	bil fa	CPl	5 weeks ao	yes	r of tension cavity	no	no	tuberculous	no	—
447	bil fa	PPl	2 weeks ao	yes	r of tension cavity	no	yes	no	yes	Died from hemorrhage into pleural space (Fig. 5)

Bil = bilateral; unl = unilateral; ma = moderately advanced; *fa* = *far* advanced; CPl = complete pneumonolysis; PPl = partial pneumonolysis; Th = thoracoscopy; *ao* = after operation; bo = before operation; r = rupture; res adh = residual adhesions; inj to l = injury to lung.

Direct injury to the lung during operation was the cause in the remaining three patients. The spontaneous pneumothorax occurred six hours, ten hours, and five days after operation.

Summing up, spontaneous pneumothorax occurred in seven instances as a postoperative complication in 521 operations. In four patients pneumothorax could be continued, eight were followed by empyema (four mixed infections), one pneumothorax was discontinued because of effusion, and two patients died within six months after operation. Table X gives a short review of these cases.

Treatment of spontaneous pneumothorax relieved the intrapleural pressure by aspiration of air or continuous underwater drainage. In later years we have used instillations of penicillin.

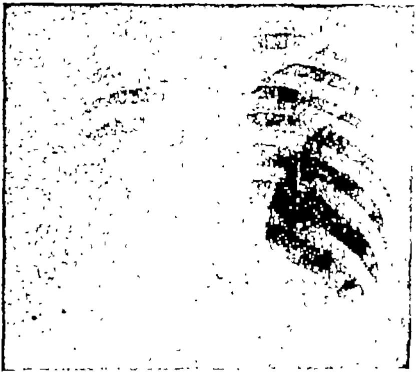

Fig. 7.—Spontaneous pneumothorax six days old, treated by closure of a persistent bronchopleural fistula.

In one of our patients we were able to close the bronchopleural fistula by endoscopic procedures. The case history is as follows:

The patient, a 30-year-old female with bilateral, far-advanced disease, was admitted to the hospital six days after the onset of a tension pneumothorax with a temperature of 104° F. Adhesions were found in the x-ray. They were considered very dangerous as removal of air at several occasions did not change the positive pressure. A thoracoscopy four days after admission visualized a shelf adhesion which was not completely cuttable. (Fig. 7.) A small bronchopleural fistula was found near to the insertion of the adhesion. It was possible to fashion a pedicle flap from the adhesion and to plug it into the small hole. Partial coagulation of the graft baked the latter to the visceral pleura with the effect of hermetic closure of the fistula. Tension pneumothorax subsided and the pressure remained negative. Contamination of the pleural cavity with *Staphlococcus aureus* and acid-fast organisms had already taken place. Onset of an empyema could not be prevented. Frequent aspiration, irrigation, and instillation of penicillin helped to dry up the empyema completely. One year afterward, the lung was completely re-expanded. No thoracoplasty was contemplated on account of the bilateral disease and a low vital capacity. Only a temporary interruption of the phrenic nerve was carried out, in order to promote the re-expansion of the lung. This patient, although having Gaffky 6 (Gaffky 9 at admission), is still alive (4 years since incident) and put on 30 lb. of weight. Later we saw an x-ray taken two months before the onset of the spontaneous pneumothorax. It revealed two interlocking giant cavities corresponding to the place where the fistula was found. X-rays and tomograms after reexpansion did not show these cavities.

Empyema.—In order to be practical, any collection of pus in the pleural cavity should be considered an empyema, whether or not organisms can be found. With a few exceptions, the nature of the empyema can be ascertained from the nature of the pulmonary lesion.

In our series of 500 cases (521 sides, 654 operations) there were seventeen instances (3.3 per cent) where an empyema developed within six months after operation without pre-existing purulent fluid. There was no case of empyema in the 21 cases with bilateral operations. Table XI shows the relationship between onset of empyema and type of operation.

TABLE XI. TYPE OF OPERATION AND ONSET OF EMPYEMA

TYPE OF OPERATION	ONSET WITHIN 4 WEEKS			4 WEEKS TO 3 MONTHS		3 TO 6 MONTHS		TOTAL
	SPONTANEOUS PNEUMOTHORAX	INJURY TO LUNG	SPREAD	SPONTANEOUS PNEUMOTHORAX	SPREAD	SPONTANEOUS PNEUMOTHORAX	SPREAD	
Complete pneumonolysis	1	0	0	0	0	1	2	4
Partial pneumonolysis	1	1	1	3	2	0	3	11
Thoracoscopy	0	0	1	0	0	0	1	2
Total	2	1	2	3	2	1	6	17

In one of these patients (No. 69) the empyema could be traced directly to a lung injury. In the two patients with spontaneous pneumothorax within the first four weeks, it was due to the rupture of a cavity. In one patient (spread of disease within four weeks) pneumothorax should never have been attempted (tuberculous bronchopneumonia with repeated severe hemoptyses).

There were four additional cases of empyema where the onset could be traced back to pre-existing lesions of the pleural cavity and where the operation did not cause it.

Acid-fast bacilli were found in sixteen cases. Five cases were mixed infections (four of them due to persistent bronchopleural fistula).

Three patients died within six months but none during the first four weeks. Table XII gives a review of all twenty-one empyema cases encountered in our series.

The accepted treatment of tuberculous empyema is a complete thoracoplasty and, in rare instances, a decortication of the lung. Thoracoplasty was performed in all those cases where the other side was free from disease or at least under control (ten cases). In mixed infections, penicillin was used since 1943 with gratifying results: 50,000 units were instilled every other day after complete removal of the pus by special posturing (Kunstler). Thoracotomy and open or closed drainage had to be done in two instances.

Adhesive Pleuritis.—Adhesive pleuritis may bring the most promising artificial pneumothorax to a sudden end. Maxwell, in a detailed study of readhesions, divides the postoperative obliteration of the pleural space into three groups: (1) acute obliteration within forty-eight hours, often total; (2) secondary obliteration which takes place within a week or two. It is more localized with predilection of the posterior mediastinum and the scapular zone. Faulty operative posture and inadequate control of pressures are claimed to be important causal factors; (3) late obliteration. It is due to pleural effusion and re-expansion of the lung with disappearance of the fluid.

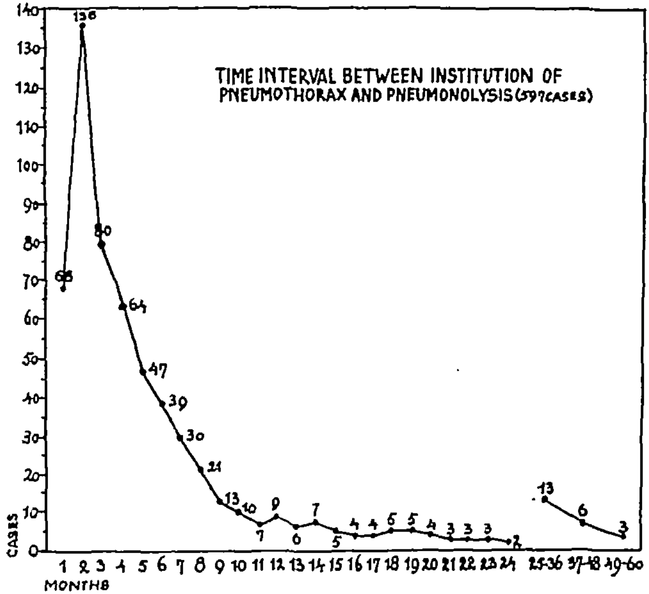

Fig. 8.—Time interval between institution of pneumothorax and pneumolysis (597 cases).

Maxwell found the incidence of readhesions in the literature 1.3 per cent while Moore calculated in base of his review only 0.29 per cent. In our cases we had seventeen cases of readhesions within the first month (0.9 per cent) and the pneumothorax had to be abandoned. In one case we tried to cut the adhesions four times, but according to the operation record the adhesions grew faster than the surgeon could cut them. In some of the seventeen cases we can attribute the failure to our negligence or the patient's, in that the refills

TABLE XII. EMPYEMATA WITHIN SIX MONTHS AFTER PNEUMONOLYSIS OR THORACOSCOPY (21 CASES)

CASE	EXTENT OF DISEASE	OPERATION	ONSET	CAUSE	BACT. OF FL. ACID F.	BACT. OF FL. MIXED	SPUT. CONV.	LUNG REEXPANDED	DIED (6MO.)	REMARKS
10	bil fa	PPl 2st	4 weeks ao	spread pleura	+	–	bil	no	yes	Tb bronchopneumonia, severe hemoptysis, not controlled by pneumothorax
16	bil fa	PPl	4 months ao	spr. to pleura	–	–	bil	no	no	
21	unl fa	Th	6 months ao	spr. to pleura	–	–	3 months ao	no	no	Phrenic nerve interruption
26	bil ma	PPl 2st	2 months ao	r. res. adh.	–	–	4 months ao	no	no	
29	bil fa	Th 3 rd Stage	2 weeks ao	Infection of pleura bo	+	–	2 weeks after 1st st	?	no	Moderate fluid bo. 3rd stage only Th.: lung covered with fibrin after first stage left against advice after last stage.
59	bil fa	PPl	2 months ao	?	+	–	bil	?	no	No sufficient record
63	bil fa	Th	2 months ao	spr. t. pleura	+	–	6 months ao	no	no	Had exudative pleuritis before operation. Thoracoplasty
64	bil fa	CPl	6 months ao	spr. t. pleura	+	–	neg. bo	yes	no	
66	unl ma	Th	3 weeks ao	spr. t. pleura	–	–	neg. bo	no	no	Had thickened pleura before operation, minimal fluid
69	unl fa	PPl	2 weeks ao	inj. t. lung	+	–	no	no	yes	Cavity collapsed, refused thoracoplasty. Fluid before operation
71	bil fa	PPl	8 weeks ao	sp. px. 6 w. ao	+	+	bil	no	no	Rupture residual adhesions. Thoracotomy, Thoracoplasty.
74	bil fa	PPl	3 weeks ao	sp. px, br-plf.	+	–	bil	no	no	Fluid before operation
78	bil fa	CPl	2 months ao	sp. px. 6 d. ao	+	+	bil	no	no	Rupture of cavity. Refused thoracoplasty.
94	unl fa	CPl	3 months ao	spr. t. pleura	+	–	5 months ao	no	no	
104	unl fa	PPl 2st	2 months ao	spr. t. pleura	–	–	1 week a. 2nd st.	no	no	Minimal fluid before operation. Thoracoplasty at 6 months.
108	bil fa	PPl 2st	3 months ao	spr. t. pleura	+	+	neg. after 2nd st.	yes	no	Penicillin local, thoracoplasty.
196	bil fa	PPl	10 days bo	sp. px, br-pl. fist. r. cav.	+	+	bil	yes	no	Closure of fistula by endoscopic operation, phrenic nerve interruption, penicillin into pleural cavity.
217	bil fa	PPl	3 months ao	spr. t. pleura	+	–	bil	yes	yes	Exudative pleuritis, 102 temperature bo. Intest. tb., poor risk.
358	unl ma	Th	before Th.	r. adhesions	+	–	6 months ao	no	no	Lesion seen at thoracoscopy. Thoracoplasty. Pneumothorax 10 ms. duration bo
429	unl ma	PPl	5 weeks ao	sp. px. r. cav.	+	+	neg. bo	no	no	
442	bil fa	CPl	3 months ao	sp. px. r. cav.	+	–	yes	no	no	Pneumothorax discontinued before rupture of tension cavity

bil = bilateral; unl = unilateral; ma = moderately advanced; fa = far advanced; CPl = complete pneumonoylsis; PPl = partial pneumonolysis; Th = thoracoscopy; ao = after operation; bo = before operation; r = rupture; res. adh. = residual adhesions; inj. to l. = injury to lung; sp. px. = spontaneous pneumothorax; br-pl. fist. = bronchopleural fistula.

were not given at the right interval or in right amounts. Wrong posturing may favour readhesions, but, as after thoracoplastics, we paid more attention to the controlateral "good" side and ordered posturing accordingly. In the majority of our cases, however, the re-expansion was due to the pathological process and could not be avoided. Most of these patients had subsequent thoracoplasty.

MORTALITY

Of the 500 consecutive patients considered in the postoperative phase, three died within four weeks after operation and five died within six months. They all had partial pneumonolysis performed and are considered in detail in Table XIII.

TABLE XIII. FATAL CASES WITHIN SIX MONTHS AFTER THORACOSCOPY AND PNEUMONOLYSIS

CASE	EXTENT OF DISEASE	OPERATION	TIME OF DEATH	IMMEDIATE AND REMOTE CAUSE OF DEATH
10	bil fa bronchopneumonia, cavities*	PPl 2 stages	2 months after operation 2nd stage	General downhill course, Gaffky VIII; spread to pleura; empyema
25	bil fa cavity r. base, phrenic paralysis	PPl 2 stages	4 months after operation 2nd stage	Pneumonectomy in 1941, died postoperatively.
69	unl fa cavity fluid	PPl	3 months after operation	Injury to lung during operation, empyema, refused thoracoplasty
138	bil fa cavities; 9 rib thoracoplasty on opposite side; Positive sputum, hemoptysis from pneumothorax side	PPl	2 weeks after operation	Pneumonitis, atelectasis, anoxemia; bronchoscopic aspirations without effect.
217	bil fa, intestinal tb; cavities	PPl	3 months after operation	Spread to pleura, empyema; general downhill course.
247	bil fa cavities	PPl	18 days after operation	Rupture of residual adhesions, spontaneous pneumothorax, bronchopleural fistula.
310	bil fa fluid, cavities	PPl 2 stages	2 months after operation	General downhill course, Gaffky VIII.
447	bil fa cavities	PPl	2 weeks after operation	Rupture of tension cavity, terminal hemorrhage from cavity into pleural space. Spontaneous pneumothorax and bronchopleural fistula (Fig. 5).

*See footnotes on abbreviations on Tables X and XII.

Only four cases can be directly attributed to the operation, which gives a six-month case mortality of 0.8 per cent and a stage mortality of 0.6 per cent.

One of these four patients with far-advanced bilateral disease had a nine-rib thoracoplasty for cavitary lesion on one side. The operation resulted in a complete closure of the cavity which was confirmed by tomogram. A pneumothorax was instituted on the other side in order to control the positive sputum and severe hemoptyses. Adhesions did not allow a selective collapse and partial pneumonolysis was performed. The patient died from anoxemia due to pneumonitis and partial atelectasis unrelieved by repeated bronchoscopic aspirations, two weeks after operation.

The second patient died from empyema three months after operation. The empyema was due to direct injury to the lung during partial pneumonolysis.

The third patient died after eighteen days of anoxemia due to uncontrollable spontaneous pneumothorax caused by rupture of residual adhesion.

The last one had a terminal hemorrhage from a blow out of a cavity two weeks after partial pneumonolysis (Fig. 5).

In the other four patients the pneumonolysis had no connection with the death. Three of them died from further spread of disease and one after pneumonectomy (1941) four months after an unsuccessful pneumonolysis.

Table XIV gives the percentage of case and stage mortality.

TABLE XIV. CASE AND STAGE MORTALITY WITHIN SIX MONTHS

	STAGE MORTALITY (%)	CASE MORTALITY (%)
All cases	1.2	1.6
Deaths due to operation	0.6	0.8

COMMENT

G. R. Howell considered a consecutive number of pneumothorax cases of the Royal Edward Laurentian Hospital and came to the conclusion that out of every 100 attempted pneumothoraces only sixteen showed a perfect result without evident adhesion. In 19 per cent no free pleural space was found and in 13 per cent the lung was adherent to the chest wall over such a broad area that the pneumothorax was immediately abandoned. In 52 per cent of the cases adhesions were found, but the pneumothorax was continued for various lengths of time. It is this group of patients that came to thoracoscopy and pneumonolysis.

While in earlier years not all of Howell's 52 per cent group came up for thoracoscopy, we later followed the policy of attempting pneumonolysis in every case in this group. We cannot exclude the presence or absence of adhesions by x-ray alone. We therefore operated on patients in the absence of radiologically demonstrable adhesions when (1) fluid covered the costophrenic angle (56 per cent of all cases had fluid from minimal to large amounts previous to operation), (2) cavities did not collapse, and sputum remained positive in unilateral cases, (3) repeated hemoptyses with the other side free from disease or under control, (4) spontaneous pneumothorax with uncontrollable bronchopleural fistula.

If a cavity remains open in the absence of adhesions or after complete pneumonolysis, it should be suspected of being a tension cavity. In such cases we abandoned the pneumothorax immediately by withdrawing air or by oxygen lavage.*

Pneumothoraces which develop fluid and high fever should also be immediately abandoned, if the temperature does not subside within a week after operation. They are potential candidates for an empyema.

75.2 per cent of our patients had a satisfactory result. Complete and partial pneumonolysis was performed on them at approximately the same rate (36.7 per cent and 34 per cent). Of the 24.8 per cent unsatisfactory cases, however, partial pneumonolysis forms the major part, with 17.3 per cent. The problem starts with those patients on whom a complete pneumonolysis cannot be done in one stage. In cases with mild, moderately, or far-advanced unilateral disease, we prefer to abandon the pneumothorax (and do thoracoplasty, lobectomy, pneumonectomy), if a complete pneumonolysis or a safe partial pneumonolysis cannot be *done in one stage.*

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*See illustrations in Surg., Gynec. & Obst. 78: 254, 1944.

There remains the group of cases with far-advanced bilateral disease. In spite of greater risks, pneumonolysis may be the only chance to achieve a satisfactory collapse. The number of patients with far-advanced bilateral conditions coming up for pneumonolysis increased rapidly in the ten-year period, while the mild and moderately advanced cases showed an increase to a much lesser extent.

Table XV illustrates the higher incidence of complications in the far-advanced and bilateral groups.

TABLE XV. MAJOR COMPLICATIONS IN RELATION TO STAGE AND DISEASE

COMPLICATION*	UNILATERAL (%)	BILATERAL (%)	MILD AND MODERATELY ADVANCED (%)	FAR ADVANCED (%)
Spontaneous pneumothorax	28.5	71.5	35.7	64.3
Empyema	33.3	66.7	19.1	80.0

*Within six months after operation.

It is somewhat difficult to appreciate the risk connected with the multistage operations. Tables IV and V show that the satisfactory results in the multistage operations are almost equal in proportion to those with a single stage operation. The surgical interference is apparently of minor importance and the great risk is rather represented by the waiting period from one stage to the other. Quite a number of residual adhesions ruptured in the interval.

We may therefore say that multistage operations should be considered only, if residual adhesions are considered safe, if the patient remains under close supervision during the interval, and if intrapleural pressures are well *adjusted and "stretching" strictly avoided.*

We may here also discuss the question of the time interval between institution of artificial pneumothorax and the operation. This was the subject of the presidential address given by J. E. H. Roberts at the meeting of the British Tuberculosis Association in 1944. He pointed out that thoracoscopy should be performed as soon as adhesions are diagnosed, or, in other words, no definite time period should be stated.

We agree with Mr. Roberts. We try to operate as soon as direct or indirect diagnosis of adhesions is made. Fig. 8 illustrates that most of our adhesions were divided within the second month after institution of pneumothorax. The shortest time between initial pneumothorax and thoracoscopy was ten days, the longest, five years.

There is a widespread opinion that within the first weeks the available airspace is too small for the introduction of the instruments. It is true that even oblique x-rays may show only a small marginal zone of air although the patient is taking 300 to 500 c.c. twice weekly without considerable change of the intrapleural pressures. In these cases the lung is always collapsed in an anterior-posterior direction due to lateral or posterior adhesions with a large air space in front of the lung. Expiratory stereoscopic x-rays are very useful. Very often a shift of the anterior mediastinum is evident and significant. At rare occasion the airspace may be situated posteriorly with consequent shift of the posterior mediastinum.

CONCLUSIONS

From discussing the results in our series of 500 cases with 654 operations, the following conclusions may be drawn:

1. Every patient with pneumothorax should be closely guarded against possible complications and submitted to thoracoscopy as soon as direct or indirect signs of adhesions are detected. Indirect signs are (a) presence of fluid in costophrenic angle, (b) insufficient collapse of diseased lung tissue, cavities, and persistence of positive sputum (unilateral cases or controlateral side under control) in spite of regular and apparently sufficient refills, (c) partial shift of the mediastinum. Direct signs of adhesions may be recognized only on a later date, but expiratory x-rays, oblique stereo x-rays, tomograms, and outlining of eosophagus by radioopaque substances make it possible to establish such a diagnosis before direct signs such as indicated are available.

2. Thoracoscopy is a harmless procedure; the only potential complications which need be considered are subcutaneous emphysema and hemorrhage from the trocar puncture. Both may be reduced by technical modifications such as those outlined. General anaesthesia is of definite disadvantage and local infiltration with novocaine is the choice.

3. Adhesions should be divided if (a) they are responsible for an insufficient collapse of the diseased part of the lung, (b) they are suitable for separation without anticipation of complications as injury to lung tissue, rupture of residual adhesions and perforation of cavities or hemorrhage.

4. Adhesions of major caliber or multiple small adhesions should not be divided if they make possible a selective collapse, especially in bilateral cases.

5. Multistage operations should be attempted only if (a) no other procedure is feasible, (b) intrapleural pressures are well controlled and (c) prevailing circumstances permit a further stage at any time considered optimal for the individual case.

6. The determination of vital capacity or of the oxygen consumption of each lung separately (bronchospirometry) should be done in the presence of bilateral disease. Bronchoscopy should be performed in all cases before institution of pneumothorax, if this is possible.

7. Pneumothorax should be abandoned immediately if (a) important adhesions are not suitable for cutting and other procedures (thoracoplasty, lobectomy, pneumonectomy) possible, (b) fluid and high fever develop and persist for more than a few days, (c) conditions which predispose to the development of empyema are present, especially injury to the lung by the cautery, rupture of residual adhesions, or of a cavity and extensive active disease of the lung,

(d) oxygen consumption is below the minimum compatible with light exercise, (f) tension cavities are present.

8. The only exceptions to paragraph (7) are those bilateral far-advanced cases with severe repeated hemoptyses originating most likely from the pneumothorax side.

9. The instruments best serving the purpose are in our opinion those designed by Coryllos. The diathermic cautery may be superior to the live cautery in some aspects but is by no means the key to a successful operation.

10. How often does pneumonolysis convert a pneumothorax which is questionable or dangerous due to adhesions, into a safe satisfactory collapse of the lung? In our series with 500 cases and 654 stages conversion occurred in 75 per cent. This result raises the number of safe pneumothoraces from sixteen to fifty-five out of every 100 cases attempted, i.e., an increase of more than three times.

11. The principal errors committed in our series were (a) unplanned timing of first and subsequent Jacobaeus operations, (b) failure to abandon pneumothorax in certain cases as outlined above, (c) attempted pneumonolysis in cases with low vital capacity, active disease of the pleura, tuberculous bronchopneumonia, tension cavities, (d) inadequate control of pneumothorax after operation, (e) injury to lung during operation, (f) extraneous factors not dependent on medical and surgical competency.

(12) Favourable results from pneumonolysis are, *ceteris paribus,* dependent on the experience and the skill of the operating surgeon. Teaching of the technique is rather difficult, due to the endoscopic nature of the procedure. A binocular telescope may be helpful for demonstration of the technique to the pupil.

We have prepared a motion picture (16 mm. color), taken first on cadavers, later on the living patient, and believe that this is a partial answer to the problem of teaching.*

SUMMARY

The technical results of thoracoscopy and pneumonolysis in 500 cases with 654 stages are discussed, the advantages and risks of the operation described in detail, and suggestions made in order to prevent the relatively frequent complications. No attempt has been made in this paper to follow up these cases for more than six months, and no conclusions were drawn as to the ultimate clinical results.

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*Limited space does not allow us to go into details of the photographic technique and illustrations. They will, however, be the subject of a separate article, to be published later.

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AN UNUSUAL HAMARTOMA (SO-CALLED CHONDROMA OF THE LUNG)

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ALTHOUGH small, so-called, chondromas in the lung are occasionally encountered as incidental findings at autopsy or as accidental findings in routine x-rays of the lungs; the occurrence of relatively large, intrapulmonary, cartilaginous tumors which produce clinical symptoms is distinctly uncommon. Ordinarily, so-called chondromas vary from a few millimeters to 3 to 4 cm. in diameter, but a few tumors varying from 5 to 15 cm. in diameter have been encountered. They may be subpleural in position, may lie near the hila or elsewhere in the lung, and are firm, dense, inclined to be somewhat lobulated, sharply demarcated, and gray and glistening upon section. The smaller tumors rarely, if ever, produce clinical symptoms.

These so-called chondromas are not pure cartilaginous tumors but contain abnormal mixtures of elements normally encountered in bronchial wall. They are, therefore, more properly referred to as hamartomas[1] or hamartoma chondromatosum pulmonis.[2]

Although hamartoma of the lung is usually considered as a benign, slow-growing tumor, it is possible that malignant changes may occur. The hamartoma in this instance is unusual because of the size of the tumor, its histologic character, and the fact that it produced symptoms and a roentgenologic appearance which strongly simulated the usual bronchiogenic carcinoma.

CASE REPORT

H. E., a salesman, aged 49 years, developed an insidious cough during February, 1943. This complaint was soon accompanied by mucopurulent expectoration and moderately severe, more or less constant, pain in the left chest posteriorly. Three months later, when he first came under the care of Dr. D. H. Ballon and one of us (H. C. B.), roentgen-ray studies revealed a large, circumscribed, slightly lobulated, dense mass in the middle and posterior portions of the left chest (Fig. 1). The left diaphragm moved but little with respiration. He was twice given bronchoscopy by Dr. D. H. Ballon at the Royal Victoria Hospital. On May 19, 1943, tissue was removed from what was thought to be a papillomatous mass in the left main bronchus. Dr. T. R. Waugh reported that the tissue consisted, for the most part, of mucous gland structures and blood clot within fibrous and muscular tissue partly surmounted by a rather fragmented, pseudostratified, columnar epithelial layer. In a few places this gave way to stratified squamous cells. Small nests of mature cells of epithelial type were present just beneath the lining cells. Dr. Waugh was of the opinion that the findings did not permit of a diagnosis of malignancy. Bleeding and profuse purulent discharge precluded the taking of a biopsy during the course of the second bronchoscopic examination carried out ten days later. Previously febrile (99 to 100° F.) on June 1, 1943, he developed a tem-

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perature of 104° F. and a fluid level was noted in the left pleural cavity. The left diaphragm could no longer be visualized. There was increased hilum detail on the right. He was referred for admission to the Jewish General Hospital. The clinical diagnosis throughout was suppurating carcinoma of the left lung.

Physical Examination.—The patient, of sallow complexion, had obviously lost considerable weight and was perspiring profusely. There was decreased respiratory excursion of the left chest. The percussion note was impaired over the greater part of the left posterior chest. Over its midportion, breath sounds were diminished. There were numerous moist râles at the base of the left lung, and scattered râles at the right base. The blood pressure was 164/80.

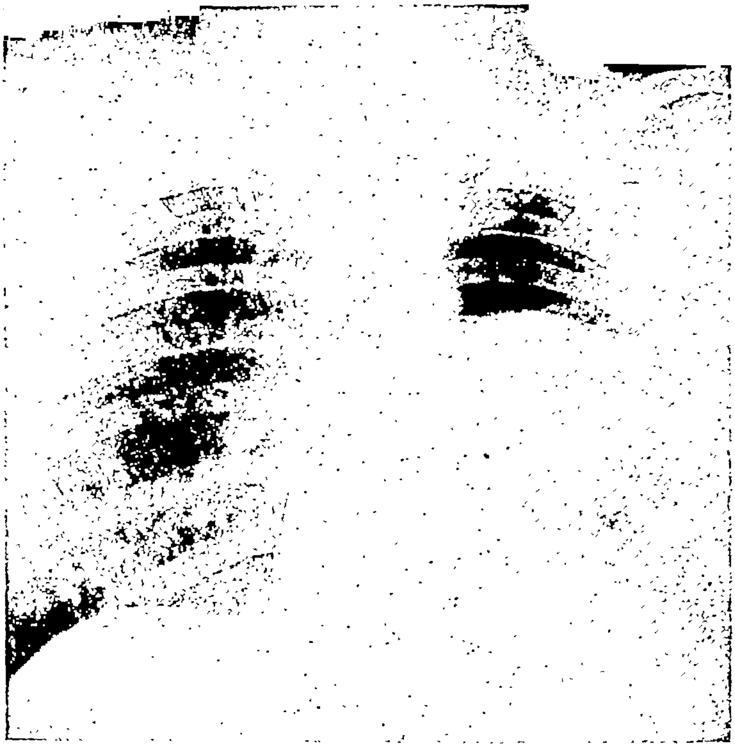

Fig. 1.—Dense, lobulated mass occupying the midportion of the left lung. The heart and mediastinal contents are drawn over slightly to the left. There is a small pleural effusion at the left base.

Laboratory Findings.—The urine showed a trace of albumin and an occasional pus cell. A hemogram showed a slight anemia; the leucocytes numbered 16,000 per cu.mm. The blood Kline reaction was negative. Repeated examinations of the sputum were negative for acid-fast bacilli. Cultures of the sputum revealed the presence of *Streptococcus hemolyticus* and *Staphylococcus aureus hemolyticus.*

Clinical Course.—A thoracentesis was performed on June 4, 1943, when a few cubic centimeters of blood-tinged fluid were aspirated. This fluid was sterile. A second aspiration on June 8 yielded a small quantity of slightly turbid, amber-colored fluid. Smears of the centrifugalized pleural fluid revealed moderate numbers of agglomerated cells, the majority of which could be recognized as polymorphonuclear leucocytes. The masses of cells showed marked degenerative changes to an extent which prevented recognition or study of their finer details. No cells could be identified as tumor cells.

Large doses of sulfathiazole did not appreciably influence either the toxic clinical manifestations or the underlying lesion. These, together with the roentgenologic evidence on

June 7, 1943, of a spill over to the contralateral lung, precluded an exploratory thoracotomy and pneumonectomy which had been contemplated. During July the patient developed hemoptysis daily. His voice became hoarse. His hemoglobin was now 56 per cent. He was subjected to a course of deep x-ray therapy by Dr. D. Berger and received 4062r without benefit.

The third and last admission was on Sept. 23, 1943, when the patient was semistuporous, unable to swallow, and had a right-sided facial weakness. His temperature was 102° F., pulse 152, and respirations were 40 to the minute. The abdominal reflexes were doubtful; there was a questionable Babinski on the right. The leucocytes numbered 14,300 per cubic millimeter. X-ray examination of the chest one day previous to his death revealed almost uniform obscuration of the left hemithorax. The cardiac shadow was observed to be shifted to the left. A left-sided, apical, paramediastinal mass was also noted. He expired on Sept. 25, 1943, eight months after the onset of his complaints.

For at least five months of this period he had exhibited signs of severe bilateral lung suppuration. His sputum, at first mucopurulent, was subsequently blood-tinged and only latterly slightly foul. The final clinical diagnosis was carcinoma of the left lung with chronic pneumonitis (abscess formation and bronchiectasis), pleural effusion, and metastases to the brain.

Necropsy Findings.—Necropsy was performed one hour and ten minutes after death.

No external abnormalities were noted. The right thoracic cavity contained a voluminous crepitant lung of a mottled gray, red, and black color. No fluid or adhesions were present on this side. Posterolaterally, the left lung was bound by dense, fibrous adhesions. The left base was adherent to the diaphragm. In the left lung a large, irregularly lobulated and globular mass of bony consistency could be palpated. This mass was continuous with a left paramediastinal mass which pulled the trachea slightly to the left. The pericardial sac contained 200 c.c. of clear fluid having a specific gravity of 1.005.

The two lobes of the left lung were fused and no interlobar division could be identified either externally or on section. The left main bronchus was slightly widened and at its division into upper and lower lobe bronchi, it was noted that the left upper lobe bronchus was pushed medially and anteriorly by the large, bony-hard mass which lay in what was probably the left lower lobe. The lumen of the left upper lobe bronchus appeared slightly narrowed and was filled with thick, gray, odorless pus. The mucous membrane of this bronchus was gray and intact. No ulceration or erosion was noted. The left upper lobe bronchus rode over the underlying bony mass.

The lumen of the left lower lobe bronchus was narrowed to a slit by the hard, bony tumor but the mucous membrane was intact. The tumor thus impinged upon the left lower lobe bronchus (Fig. 2). The main mass of tumor, however, occupied the lateral and posterior portions of the left lower lobe and extended out to the pleura.

The bony mass could be cut by the saw only, and was extremely eburnated. On section, a roughly oval, lobulated mass of firm, gray tissue sharply demarcated from the surrounding lung was observed. A large part of the tumor, which occupied the posterior portion of the left lower lobe, lay subpleurally. This mass measured 11 by 8 cm. (Fig. 3). In places it was of bony firmness, elsewhere it was gray and translucent and varied from cartilaginous to fleshy in consistency. In other areas multiple, fine, bony spicules could be palpated. In the peripheral portion of the large mass a secondary, irregular cavity filled with debris and bony spicules measuring approximately 5 × 2.5 cm. was encountered.

The tumor extended posterior to the left major bronchus. Here it was lobulated, fleshy in consistency, and at first the mass resembled lymph nodes (*C*, Fig. 2). Section through this area revealed these masses to be continuous with the large mass described above.

In no situation did the tumor mass break into a bronchus or appear to be derived from bronchial wall. Several lymph nodes in the mediastinum showed a mottled gray and black cut surface without gross evidence of tumor invasion.

The major portion of the left upper lobe was gray, firm, and consolidated. Multiple small, irregular cavities with shaggy walls filled with pus were noted. These varied from 2 to 15 mm. in diameter. The portion thought to be left lower lobe, which lay below the tumor mass, was noncrepitant, gray, and fibrous, and contained several irregular, bronchiectatic cavities lined by thick, hyperemic mucous membrane and varying from 1 to 2.2 cm. in diameter.

There was a brain abscess in the left cerebral hemisphere with focal fibrinopurulent meningitis, but the balance of the organs failed to show significant changes.

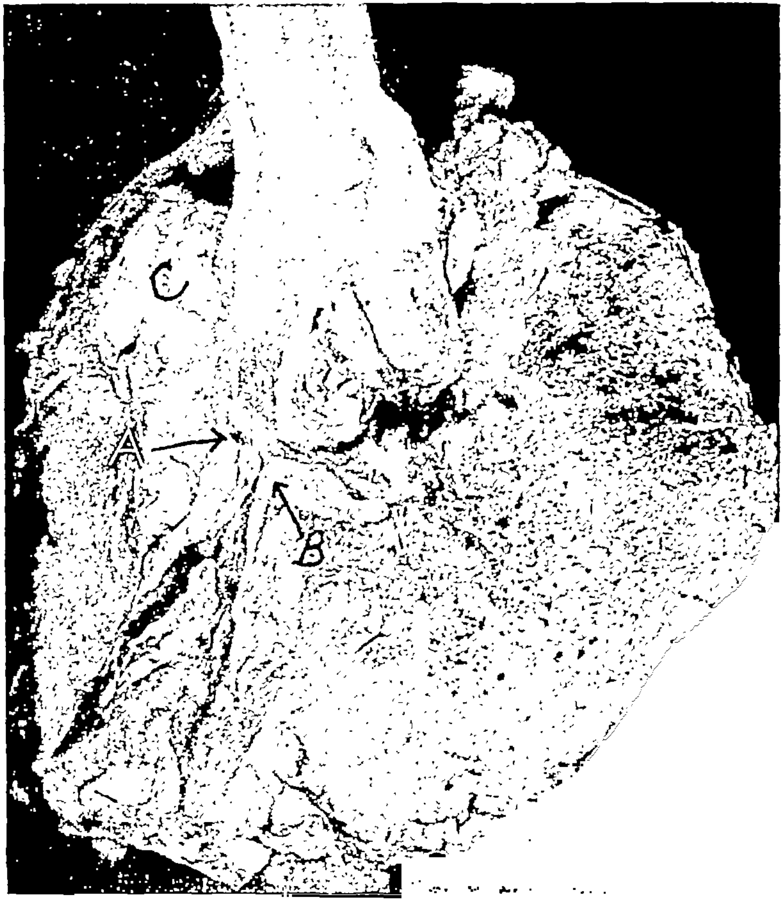

Fig. 2.—The left upper lobe bronchus, *A*, has been narrowed. The lower lobe bronchus, *B*, is slitlike. *C*, Represents a portion of tumor.

Microscopic Findings.—Microscopically, the tumor was quite variable in composition. Sections taken from the softer and more fleshy portions of the tumor showed a richly cellular pattern in which the cells varied from fat-spindle to oval, or round. The nuclei of these cells were vesicular, varied considerably in size, shape, and staining quality, and showed not infrequent bizarre and atypical mitotic figures (Fig. 4). Between the cells there was a variable amount of poorly defined, pink-staining matrix, which at times appeared to be protoplasmic processes of cells and at other times appeared to be amorphous or fibrillar. In some situations numerous giant cells containing from two to twenty nuclei were seen scattered irregularly throughout the tissue. In a few situations some of the tissue appeared to lie in lacunar spaces, suggesting a transition to young cartilage cells.

In other portions of the fleshy tumor the cells appeared more spindlelike. Here the tumor was slightly whorled and richly cellular and occasional mitotic figures were encountered.

Here and there small islands of fibrocartilage were seen (Fig. 5) and the tumor was well vascularized by thin-walled blood vessels, some of which contained hyaline thrombi.

Sections taken through the fleshy tissue in which fine bony spicules were felt showed a richly cellular pattern in which the cells were distinctly spindle-shaped and uniform in size and shape. Here numerous irregular islands of hyaline and fibrocartilage showing various stages of calcification were seen. Some areas were encountered where cells were polymorphic and atypical and near the periphery of one nodule the mediastinal fat was invaded. All types of transition from spindle cells to cartilage were noted.

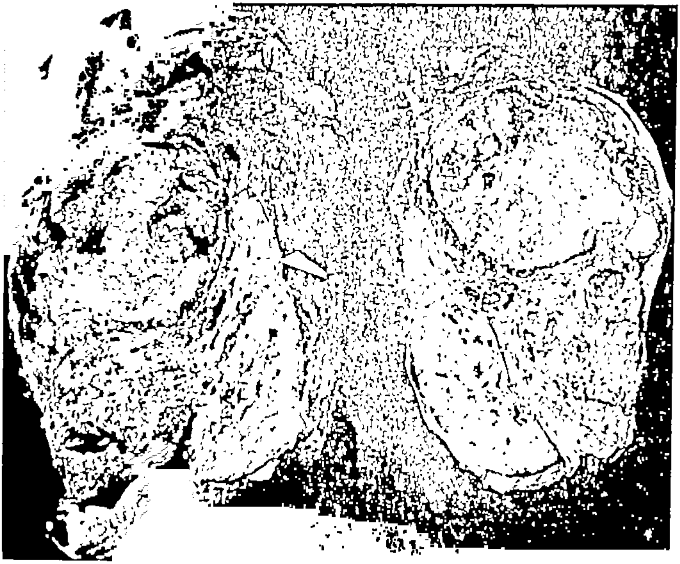

Fig. 3.—Saggital section through left lung showing tumor with secondary cavity and adjacent nodules. Note the organizing pneumonia and bronchiectasis of the left upper lobe.

Sections taken from the bony mass which required decalcification showed a rather cellular fibrous stroma in which numerous islands of cartilage in various stages of calcification were seen. In some places these calcified masses of cartilage branched and anastomosed to form typical bony trabeculae which contained Haversian canals. No fatty marrow was encountered and no foci of hemotopoiesis were seen. Between the bony trabeculae and in the cellular portion several irregular and elongated spaces were encountered which were lined by single layers of low columnar to cuboidal, ciliated epithelium (Fig. 6). This epithelium rested directly upon the cellular tumor stroma and contained no recognizable muscle or glands (Fig. 7). In the periphery of this section the capsule of the tumor was absent and tumor with bony spicules was found invading the compressed, adjacent lung and surrounding small bronchioles (Fig. 8).

Section through the left upper lobe bronchus where the tumor impinged upon the bronchial wall and narrowed the lumen showed a richly cellular spindle-shaped mass of tumor cells in which spicules of bone were present. The tumor lay adjacent to but did not involve or appear to be derived from the bronchial cartilage or perichondrium. Adjacent lymph nodes showed no metastatic tumor (Fig. 9).

Sections taken to include the secondary cavity within the tumor mass showed extensive bland necrosis of the cellular matrix with only a few bony and calcified cartilaginous trabeculae remaining.

To summarize, the tumor is large and is intrapulmonary in position. It is roughly lobulated, sharply demarcated, and as far as can be determined, does not arise from bronchial wall. This tumor showed every stage of bone genesis from a primitive osteoid tissue passing through cartilage formation to calcification. In one situation there is included deep within the bony portion of the tumor several structures lined, in part, by ciliated epithelium which, in all probability, are integral parts of the tumor rather than residual lung structures surrounded by invading tumor. In many places, from a histologic point of view, the tumor is frankly benign. In other situations malignancy cannot be excluded upon objective histologic grounds. In one area mediastinal fat has been invaded and in another the capsule of the tumor is absent and invasion of lung parenchyma is seen.

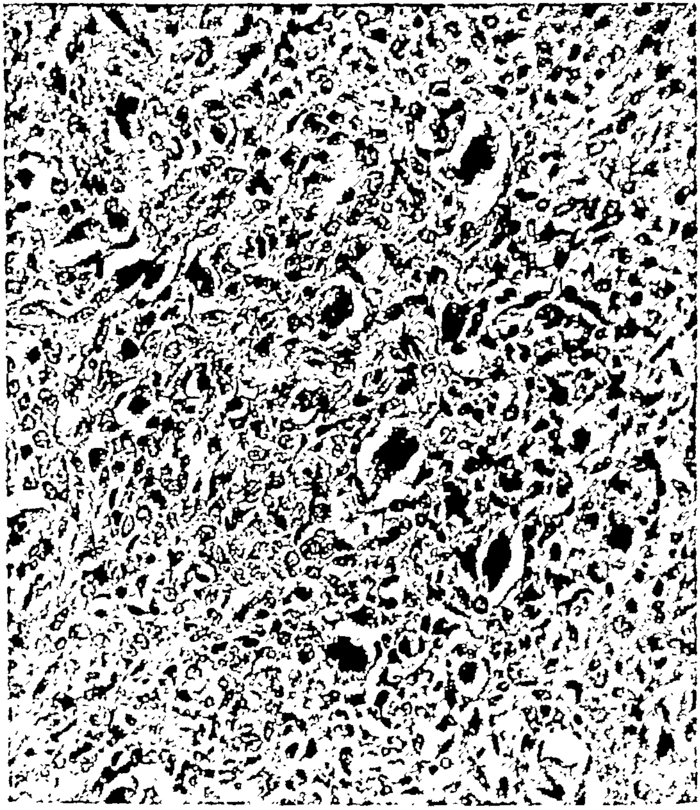

Fig. 4.—Cellular polymorphic area containing numerous giant cells. In this area, mitotic figures were frequent and malignancy could not be excluded upon objective histologic grounds.

DISCUSSION

What is the nature and genesis of this tumor? There is little doubt that this tumor is primary in origin in the lung. The remote possibility that this tumor represents a metastasis from a small, cryptic, primary malignant bone tumor cannot be seriously considered. The absence of other symptoms and signs, the solitary nature of the tumor, its partial encapsulation and lack of metastases elsewhere preclude such a possibility.

This tumor does not appear to be connected with or arise from any existing cartilaginous rings, although the bronchus rides over it. The tumor lies within the lung substance, which it has pushed aside, and largely beneath the pleura. It encroaches upon the hilum. In this respect the present tumor is similar in position to those so-called chondromas of the lung collected and reported by Hicky and Simpson,[3] Verga,[4] Benninghoven and Pierce[5] and McDonald, Harrington, and Clagett.[6] All the above authors have noted the close proximity of these tumors to the pleura. In none of their cases did the tumor appear to arise in the mediastinum and extend secondarily into the lung parenchyma.

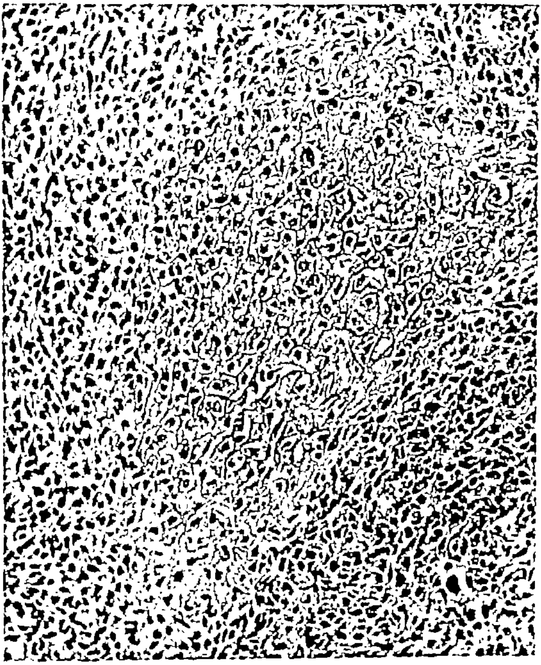

Fig. 5.—Area of fibrocartilage developing in cellular polymorphic portion of tumor.

The fact that the present tumor contains bone and shows transitions from cellular *spindle-cell matrix* through cartilage, calcification, and ossification, does not necessarily argue its derivation from osseous tissue, whether arising from a focus of osseous metaplasia in a scar, from a healed tuberculous lesion, or from a focus of bony metaplasia in a cartilaginous ring, for the so-called chondromas of the lung which have been reported in the literature have shown various stages of calcification and ossification and, indeed, such changes might readily be expected. The majority of investigators describe their tumors as having a rocklike consistency. The possibility that this tumor arose on the basis of bony metaplasia which has been described in Henke-Lubarsch[7] as occurring in chronic organizing pneumonia seems unlikely, for these bony spicules are small, needlelike, widely dispersed, and never tumorous in character, and no transitions between cartilage and cellular matrix are observed such as occurred in this tumor.

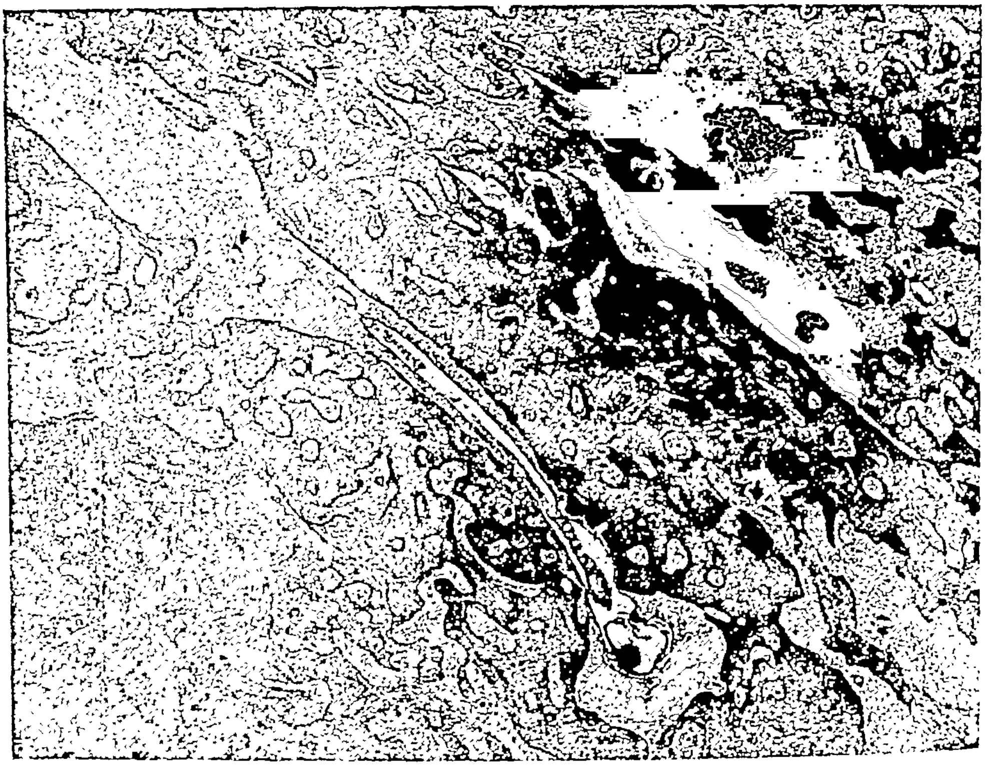

Fig. 6.—Low-power view of frankly bony portion of tumor with osteoid matrix. In the center is a long slender space lined by cuboidal epithelial cells, some of which are ciliated.

The findings of irregular spaces deep within the tumor which are lined by epithelium, some of which is ciliated, are, perhaps, of greatest significance in attempting to arrive at an understanding of the genesis of this tumor. The fact that these spaces are composed only of a layer of epithelial cells resting upon the cellular matrix of the tumor without other recognizable components of bronchial or bronchiolar wall indicates that these structures are an integral part of the tumor rather than remnants of bronchial structures which were surrounded and isolated by invading tumor.

In the great majority of the so-called chondromas of the lung reported in the literature, various admixtures of glandular spaces, lined at times by ciliated and nonciliated epithelium with or without papillary infolding, fat, blood vessels, smooth muscle, and occasionally lymphocytes, have been noted. Various names such as "adeno-fibro-lipo-chondromyxomatosum" "lipo-chondro-adenoma," "adeno-chondroma'' and ''lipo-chondroma'' have been applied to these tumors. Bayer[8] reported five cases of what he called bronchial hamartoma and Goldsworthy[3] reported a case which he labeled hamartoma chondromatosum pulmonis.

The presence, therefore, of both mesenchymal and ectodermal elements in the above-mentioned and in the presently discussed tumor suggests that we are dealing with a hamartoma or teratoma. According to Albrecht,[1] who first used the term, a ''hamartoma (from the Greek, meaning to fail or err) is a tumorlike malformation in which occurs actually only an abnormal mixing of the normal components of the organ. The abnormality may take the form of a change in quantity, arrangement, or degree of differentiation, or may comprise all three. The deduction to be drawn from the histologic examination of these formations is that they have originated in an abnormal mixing of the elements or from disturbance of their development.''

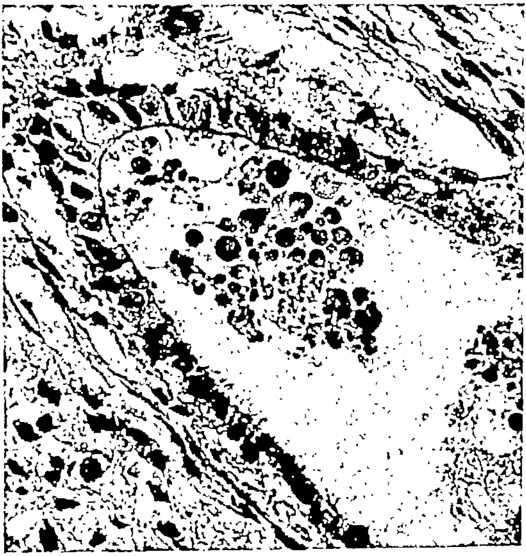

Fig. 7.—High-power view of space lined by ciliated epithelium. Note absence of muscle, glands, and note that the epithelial cells rest directly upon osteoid matrix.

In the tumor, in our case, the presence of cartilage and of spaces lined by ciliated epithelium can be regarded as an abnormal mixing of normal components of the bronchus. The quantities of these elements and their degree of differentiation do not invalidate the definition. Indeed, Borst,[9] suggests that such developmental disturbances may become the seat of growth excesses and may become truly neoplastic or even malignant in character. Borst[9] also noted that cartilage occurs in a large proportion of hamartomas and suggests that the cartilage present in many of the chondromas of internal organs was due, in part, to metaplasia of connective tissues and, in part, to embryonic scattering of cartilaginous germ centers. Mallory[10] has also stressed the importance of metaplasia in cartilage-containing tumors of soft parts. In the present case, areas were encountered in which dedifferentiation was quite marked and where the tumor was definitely neoplastic if not malignant in character.

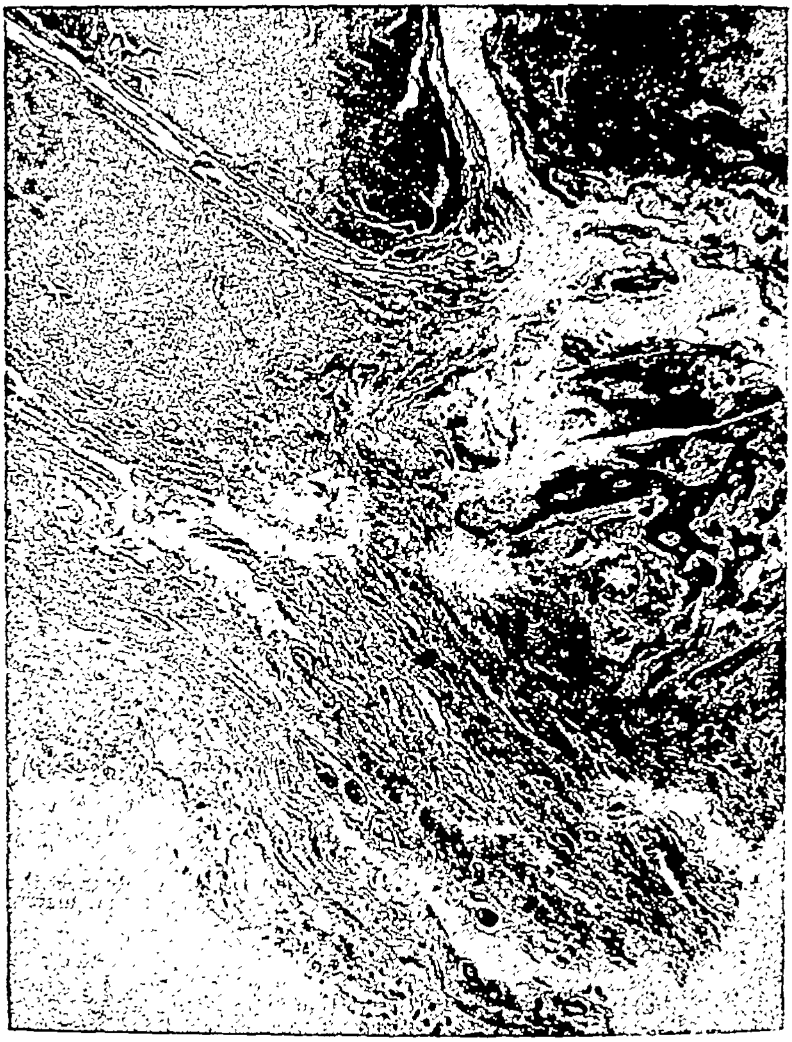

Fig. 8.—Compressed peripheral portion of lung (lower left) showing lack of capsule of tumor. Note that the tumor surrounds an intact bronchiole on the right.

The term hamartoma is all-inclusive and does not necessarily refer to a given organ or tissue overgrowth type. Therefore, with Goldsworthy,[2] who suggested the name of "hamartoma chondromatosum pulmonis," the authors would agree that specific localization and description might be included in the term and, to this end, one might call the present tumor "hamartoma osteochondromatosum pulmonis malignum."

There are those who may possibly wish to employ the term "mixed tumor" in the sense of Womack and Graham.[11] For those who wish to regard this tumor as a teratoma in which there has been an overgrowth of one element with suppression of the others, the following objections may be raised. No other structures than those normally found in this region were discovered in this tumor. The present tumor, unlike the majority of teratomas, does not arise anatomically from the mediastinum.

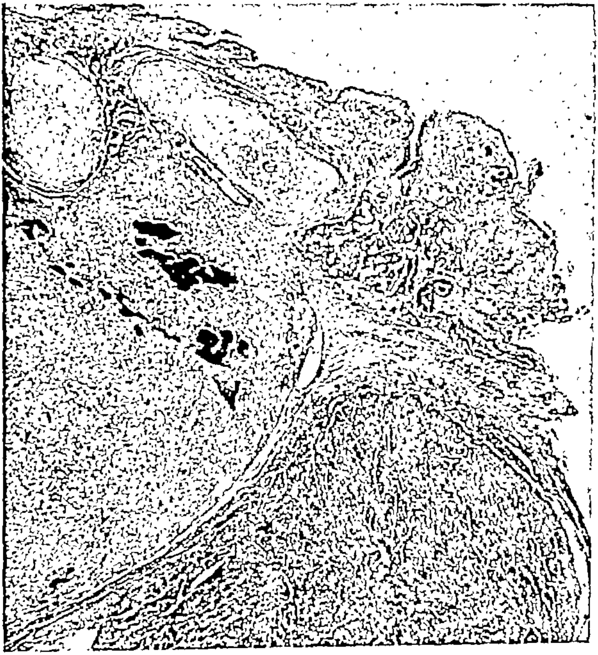

Fig. 9.—Left upper lobe bronchus passing over tumor. Note cellular structure of tumor, left, containing bony spicules. The lymph node on the right is not invaded. Note intact perichondrium about bronchial cartilages.

Although, from a clinical point of view and in the absence of metastases, this tumor may be regarded as benign, it is apparent from an objective histologic point of view that malignancy cannot be excluded. Furthermore, evidence of lack of capsule and local invasion in the periphery of the tumor is present. This recalls the unique case of a pulmonary osteochondrosarcoma reported by Greenspan[12] which showed locally invasive features. It is probable that the origin of his reported tumor was from a hamartoma, although no epithelial-lined structures or other tissue types were noted. From a purely theoretical point of view, there is no valid reason why a hamartoma or any other congenital malformation may not, under certain conditions, show malignant changes.

From a clinical point of view our case is of interest in that it may be assumed that the tumor had been present for many years. When it impinged upon the narrowed major bronchi it produced signs not unlike those associated with bronchial obstruction due to carcinoma. The original and all subsequent x-rays were suggestive of tumor but failed to reveal the characteristic findings noted in the x-ray of the specimen. Most so-called pulmonary chondromas are silent because of their usually small size and intraparenchymal position. In Klages'[13] case, the first in which the diagnosis was made roentgenologically and confirmed by histological examination of the specimen, the patient was free of symptoms. Similarly, in one of the two cases reported by Benninghoven and Pierce,[5] which was treated surgically, no symptoms were present. In only one of the three cases reported by McDonald, Harrington and Clagett[6] in which surgical removal was performed, were symptoms present, and these consisted of dyspnea on exertion and a burning sensation in the anterior portion of the thorax. It is to be noted that intralumenal polypoid tumors which may contain chondromatous elements such as those reported by Moore,[14] Gebauer,[15] and Davidson[16] are not included with hamartomas.

So-called pulmonary chondromas are not unique in man but have been noted in domesticated animals.[17]

SUMMARY

An unusually large hamartoma (so-called chondroma) of the lung is reported. It was responsible for signs suggestive of bronchiogenic carcinoma. It would perhaps be preferable to describe the hamartoma in our case as a hamartoma osteochondromatosum pulmonis malignum. The tumor which we have described showed features which upon objective grounds do not permit one to exclude the possibility of malignancy.

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ADENOMAS ARISING FROM SMALL BRONCHI NOT VISIBLE BRONCHOSCOPICALLY

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BRONCHIAL adenomas usually occur in the larger bronchi and can be visualized through the bronchoscope. The diagnosis is often established by the microscopic examination of biopsy material removed endoscopically. In an extensive discussion of bronchial adenoma by Foster-Carter[1] in 1941, the statement is made: "A bronchial adenoma arises invariably in one of the larger bronchi and there is no record of this tumor occurring in the periphery of the lung." Apparently all of the adenomas reported in the literature at that time were in portions of the tracheobronchial tree that could be visualized through the bronchoscope. Moersch and associates[2] recently stated, "Adenomas invariably arise from the main stem or larger bronchi." Among the patients with bronchial adenoma that we have treated, there are five in whom the tumor was located in a branch bronchus not accessible to the bronchoscope. In each instance the tumor was large enough to be clearly demonstrable on the roentgenogram of the chest. In two patients an x-ray was taken because of thoracic symptoms. In two other cases the tumor was found during an x-ray survey. In one patient the pulmonary lesion was found during a gastrointestinal roentgen study. Since the tumor did not arise from a large bronchus in any of the cases, it is understandable that none of the patients had any history or symptoms of secondary pulmonary suppuration. In four of the cases the tumor was located near the hilar area (Figs. 1, 2, 3, and 4) but arose from a small branch bronchus rather than from the main lobar bronchus. In the fifth case the tumor was at the periphery of the lung close to the diaphragm (Fig. 5). Three of the patients were asymptomatic, one patient had had several hemoptyses, and one had slight pain in the chest. Lobectomy was done in four cases and partial lobectomy performed for the peripheral tumor in the fifth case. All patients had an uncomplicated postoperative course and are in good health at this time, two years eight months to six months after operation.

In the four cases in which the bronchial adenoma was located near the hilar portion of the lung, a correct diagnosis was made at the time of operation on the basis of the gross characteristics of the tumor. Although the peripherally located tumor had a similar consistency, its most unusual location made us hesitate to diagnose an adenoma until that diagnosis was demonstrated by pathological examination (Fig. 6).

It is important that the thoracic surgeon recognize the gross characteristics of bronchial adenoma. The tumor is moderately soft, being considerably softer than the typical carcinoma. Not infrequently some lobulation is present. The

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tumor is usually reddish-brown in color. If a portion of the tumor projects into the mediastinum, small veins are often seen crossing over the surface. In most instances a portion of the tumor can be enucleated from the adjacent pulmonary tissue or mediastinal tissues, but there is no cleavage plane in the region of the bronchus of origin. If the tumor projects into an interlobar fissure, it may be noted that when the pleura is divided over the tumor, that portion of the tumor which projects toward the adjacent lobe can be readily freed, so that lobectomy is feasible. We do not feel that pneumonectomy is necessary in cases of bronchial adenoma that are typical and so located that the tumor can be completely removed by lobectomy. None of our patients had involvement of the regional lymph nodes.

CASE REPORTS

CASE 1.—G. H., male, aged 40, was admitted to Memorial Hospital on Dec. 6, 1943. A year previously the patient noticed slight dyspnea on exertion and fatiguability. Several hemoptyses had occurred after exertion. Physical examination was negative. Roentgenogram of the chest revealed an elongated, well-demarcated shadow extending out from the left hilum. Associated with this mass there was a moderate increase in density of the anteromedial portion of the left upper lobe suggesting a partial atelectasis. The remaining lung fields were clear. The roentgenographic findings indicated a tumor at the left hilum, probably arising in the region of the ventral segment of the left upper lobe (Fig. 1). Bronchoscopy was negative.

At operation on Dec. 7, 1943, a well-encapsulated ovoid lesion which lay in close proximity to the main stem bronchus and hilum was found. A dissection type of left upper lobe lobectomy was performed. On examination of the surgical specimen, a small portion of the lobe was found to be dark in color, moderately firm in consistency, and apparently airless; the remainder was aerated. Involving the hilar region and situated just adjacent to the main lobar bronchus was an irregular, ovoid-shaped tumor. It was pink in color, showed some lobulation, and was apparently encapsulated and well demarcated from the adjacent pulmonary tissue. It measured 4.5 × 3.1 × 2.3 cm. Section of the lung showed that there were three main branches of the lobar bronchus. Two were situated below the tumor and the uppermost one of these two was displaced downward and was closely associated with the tumor, but there was a definite line of cleavage between the two. The other branch of the bronchus was located above and lateral to the tumor. Microscopic diagnosis was bronchial adenoma. The patient had an uneventful postoperative course and has been well and asymptomatic since.

CASE 2.—K. I., female, aged 27, was admitted to Lenox Hill Hospital on June 26, 1944. Five years prior to admission the patient developed pain in the chest and an x-ray revealed a mass in the left lung. Repeated roentgenograms since then had shown gradual enlargement of the mass, although the patient had no pulmonary symptoms excepting occasional slight discomfort in the chest. On admission a chest roentgenogram revealed an oval, sharply marginated shadow of increased density at the posterior aspect of the lower portion of the left hilum, measuring about 4 cm. in diameter (Fig. 2). On June 28, 1944, a thoracotomy was performed. In the upper part of the lower lobe, bulging somewhat into the interlobar fissure, a rounded, slightly firm tumor mass was found in close association with the bronchus of the lower lobe. The mass was slightly lobulated and could be enucleated from the adjacent pulmonary tissues over a considerable area, except at its attachment to the bronchus. The lower lobe was air-containing and showed no atelectasis or signs of inflammation. A left lower lobe lobectomy of the dissection type was performed. Examination of the surgical specimen revealed that the tumor arose from a small branch bronchus adjacent to the lobar bronchus. No inflammation was present. The pathological diagnosis was bronchial adenoma. The postoperative course was uneventful and the patient has remained well.

CASE 3.—C. K., male, aged 42, was admitted to Lenox Hill Hospital on Feb. 1, 1945. One month prior to admission a routine x-ray of the chest at his place of employment showed

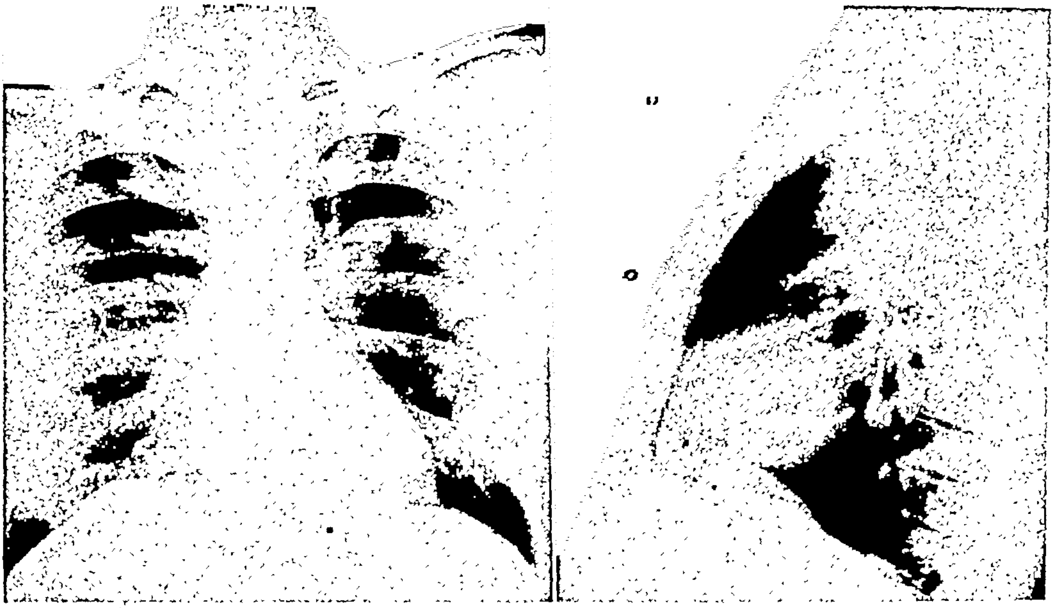

Fig. 1.—Mass in hilar region of left upper lobe.

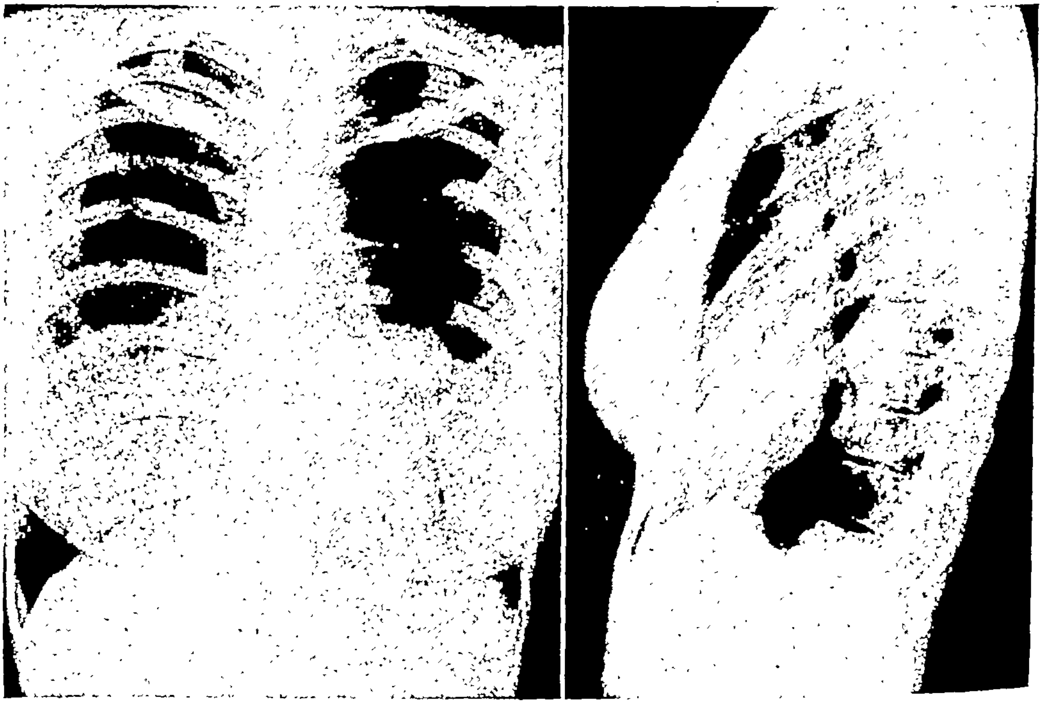

Fig. 2.—Circumscribed mass near hilum of left lower lobe.

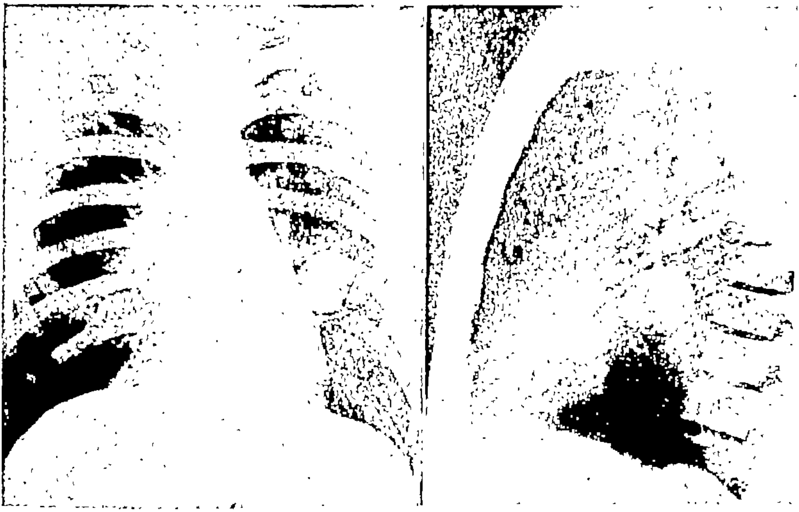

Fig. 3.—Sharply circumscribed mass near hilum of left lower lobe.

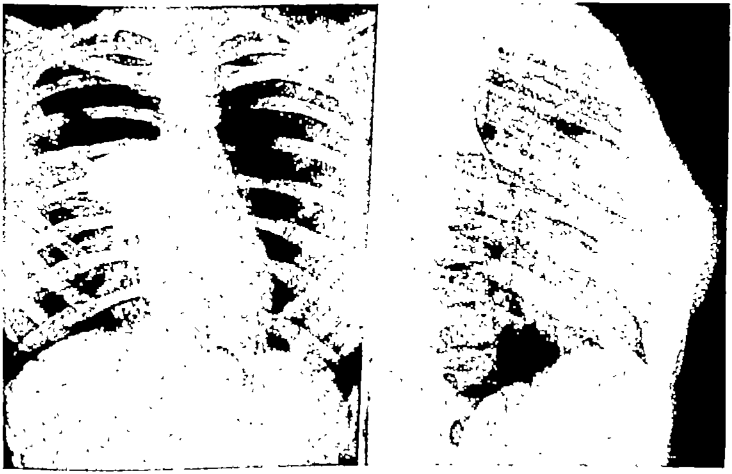

Fig. 4.—Large mass in anterior portion of right upper lobe with distal atelectasis.

a lesion of the left lung. He had had no symptoms. No previous chest x-ray had ever been taken. Physical examination was negative. Roentgenogram of the chest showed a globular shadow of increased density, measuring about 4 cm. in diameter, located in the left lower lobe adjacent to the inferior portion of the hilum (Fig. 3). At operation on Feb. 3, 1945, a mass could be palpated in the hilar portion of the left lower lobe. The tumor bulged into the interlobar fissure and encroached slightly on the upper lobe. On exposure the mass was somewhat lobulated, and a portion of it could be enucleated from the surrounding tissue. A dissection type of left lower lobe lobectomy with complete removal of the tumor was done. Examination of the gross specimen revealed that the tumor did not present in the lumen of the main lobar bronchus and there was no obstruction of this bronchus. The tumor appeared to arise from a small branch bronchus adjacent to the main bronchus. No inflammation was present. Microscopic examination of the tumor revealed a bronchial adenoma. The postoperative course was uneventful and the patient has remained well.

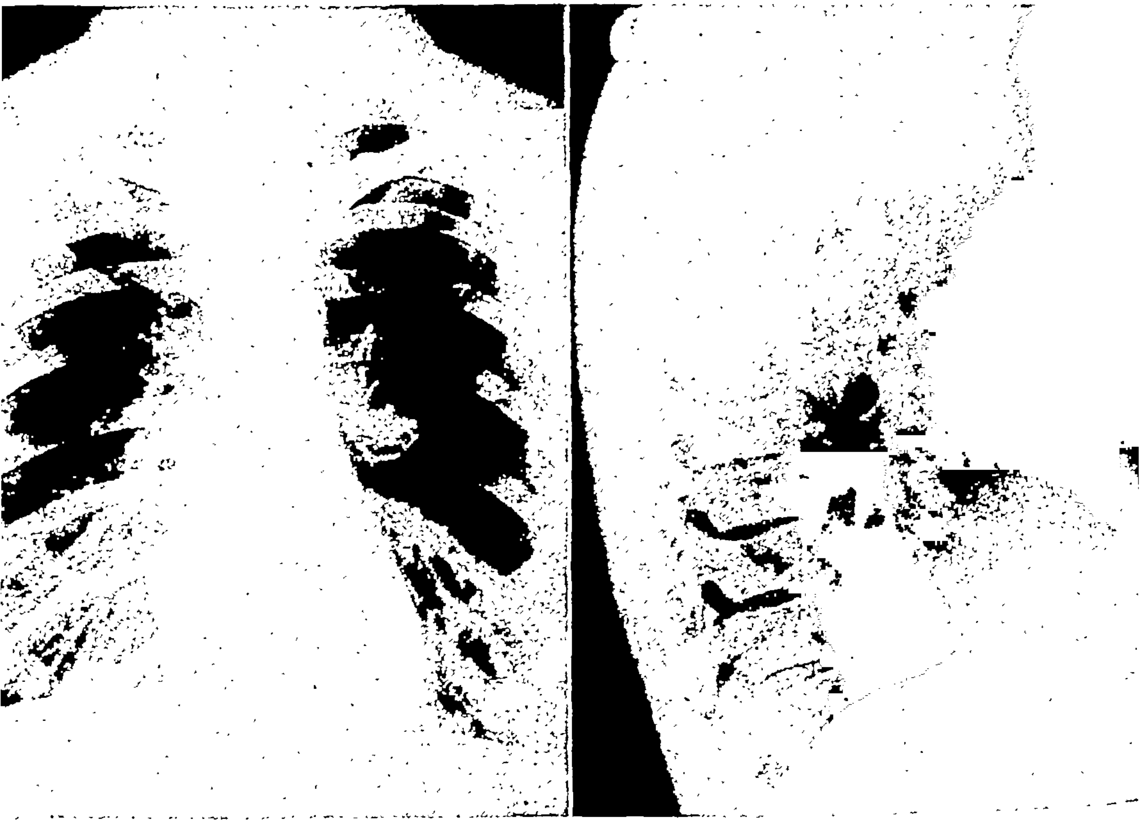

Fig. 5.—Posteroanterior film shows rounded mass projecting partly above dome of right diaphragm. Lateral film shows mass posteriorly in costovertebral portion of right lower lobe just above diaphragm.

Case 4.—I. D., Negress, aged 16, was admitted to Lenox Hill Hospital on Jan. 30, 1946. Routine roentgenogram of chest made in a tuberculosis survey in November, 1945, revealed a mass in the right middle lung field. The patient had always been in good health and had had no symptoms referable to the lungs. Physical examination was negative. Roentgenogram of the chest showed an irregularly shaped mass measuring approximately 6 cm. in its greatest dimension, situated near the root of the right lung. The lateral film localized the mass anterior to the hilum in the region of the short fissure. A small area of slightly increased density was noted in the lung field lateral to the tumor mass (Fig. 4). Bronchoscopy was negative. Thoracotomy was performed on Feb. 1, 1946. There were vascular adhesions pres-

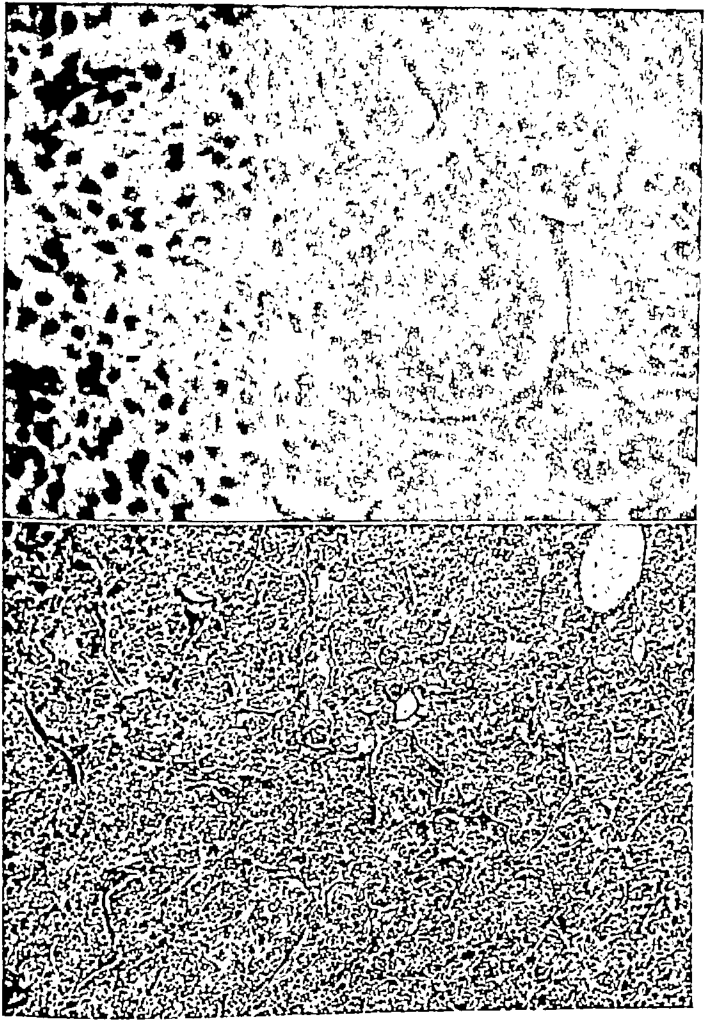

Fig. 6.—Photomicrographs of the peripherally located bronchial adenoma.

ent over the lower anterior portion of the upper lobe. This portion of the lobe was airless and had a yellowish appearance. A tumor was present near the hilum of the upper lobe. A dissection type of right upper lobe lobectomy was performed. Examination of the specimen revealed a moderately firm mass arising from the lower anterior branch bronchus. The lung peripheral to the tumor was largely airless and rubbery in consistency, and on section revealed a soft, golden-pink tissue. Microscopic examination of the tumor revealed a bronchial adenoma. Section through the golden-pink pulmonary tissue distal to the adenoma revealed large foam cells with extensive cholesterin deposits. Sections of the lymph nodes showed no tumor. The postoperative course was uneventful and the patient has remained well.

CASE 5.—M. U., female, aged 38, was first seen at Memorial Hospital in January, 1945, when she complained of epigastric pain and postprandial vomiting of seven months' duration. The vomitus had been bloody on two occasions. She had been unsuccessfully treated by diet. Roentgenograms were interpreted as showing a soft tissue mass in the lower peripheral portion of the right lower lobe (Fig. 5) and pyloric obstruction, possibly due to a peptic ulcer. An aspiration biopsy of the lung lesion was reported as probable malignant tumor. At operation on March 8, 1945, a soft, sharply circumscribed tumor mass was found in the right lower lobe just beneath the diaphragmatic and posterior pleural surfaces of the lobe. There was no atelectasis and the adjacent pulmonary tissue appeared normal. Because of the peripheral location of the tumor and the absence of any other pathology in the lobe, a wedge resection of that portion of the lung in which the mass was situated was performed, and frozen section done immediately. The microscopic diagnosis was bronchial adenoma (Fig. 6). On examination of the gross specimen, the tumor mass, measuring 5 × 5 × 4 cm. was found well circumscribed and demarcated from the surrounding pulmonary parenchyma by a fine but definite capsule. In no place did it invade or ulcerate the overlying visceral pleura. The gross appearance resembled lymphoid tissue. The patient had an uneventful postoperative course. Four months later gastric resection was performed for obstructing duodenal ulcer. The patient has been well since that time.

SUMMARY

Five cases of bronchial adenoma arising from small bronchi are reported. In each instance the adenoma was located in a portion of the bronchial tree that cannot be visualized bronchoscopically. Hemoptysis was present in only one case. At the time of operation, none of the patients had any pulmonary suppuration. Lobectomy was performed on four patients. Due to the peripheral location of the adenoma in the fifth patient, a partial lobectomy was done. All patients had an uncomplicated postoperative recovery and are well at this time.

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FASCIA LATA GRAFTS FOR CHEST WALL DEFECTS

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CHEST wall tumors comprise a group of neoplasms which has been adequately covered in the medical literature. Hedblom,[1, 2] in the year 1921, and again in 1933, reviewed all reported cases and added his personal observations. Sommer and Major[3] reviewed all references to the subject from 1933 to 1940 and added fifteen cases of their own. In these reports the authors agreed that the closure of large, surgical, chest defects was sometimes very difficult. Hedblom used various measures for producing adhesions between the lung and that part of the chest wall which was to be resected. His operations were carried out in stages. Iodoform gauze was first packed subcostally for a time before the pleura was opened or the lung itself was sutured to the chest wall. When approximation of the intercostal structures was not possible, Sommer and Major attempted airtight chest wall closure by careful suture of the extracostal muscles, the subcutaneous tissue, and skin. Effler and Blades[4] in a recent discussion of "Thoracic Centers in World War II," also stated that serious defects in the chest wall were not always readily closed with skin, muscle, and bone of the thorax. In a few isolated instances, tantalum plates have been used to bridge the defects in the chest wall, but these endeavors were commonly disastrous because the plate acted as a foreign body and either sloughed out or became loose and moved with respirations and had to be removed.

Fascia lata transplants have been or could possibly be used to serve many different purposes. Kirschner[5] in 1914 listed, among other uses for fascia lata grafts, the possibility of closing defects in the thoracic wall. In 1924, Brenner[6] used fascia lata in the reconstruction of a portion of the male urethra. In 1924, Gallie and LeMesurier[7] used free transplants of fascia lata as living sutures in the treatment of hernia. During World War II, fascia lata transplants were often used in repairing large dural defects,[8] to stop the loss of cerebrospinal fluid and prevent the entrance of organisms through the meningeal defect. In 1930, Zinninger[9] reviewed tumors of the chest wall at the Peiping Union Medical College Hospital. He presented one case of a 23-year-old Chinese carpenter who had a fibrosarcoma of the right epigastrium. It was necessary to excise the entire thickness of the abdominal wall in order to remove the tumor and the peritoneal cavity had to be opened. In closing the wound it was possible to approximate the skin and subcutaneous tissues, but there remained a defect in the peritoneum, muscle, and fascia about the size of a man's hand. This he closed with a layer of fascia lata from the right thigh, fixing it with interrupted silk sutures to the peritoneum and fascia on both sides. The skin flaps were then closed over the fascial transplant.

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It is the purpose of this paper to present four cases of large chest wall tumors in young patients which were resected, and the resultant surgical defects successfully closed with the aid of large, wide, free, fascia lata grafts. The procedure seems to be a practical one, which probably can be used more frequently in the future.

TECHNIQUE

Endotracheal gas, oxygen, and ether anesthesia is employed and the patient is prepared as for any other major chest operation, but, in addition, one thigh is also prepared and draped. After a wide resection of the tumor-bearing portion of the chest wall, a fascia lata graft, the same size as the defect, is taken from the selected thigh (Fig. 1), and the "shiny side," that is, the surface adjacent to the subcutaneous tissue, is placed so as to be in contact with pleura. It is then secured to the parietal pleura and periosteum with multiple interrupted silk sutures as shown in the diagram in Fig. 2. A #18 French catheter is left in the pleural space until the chest is completely closed in order to facilitate the removal of all free pleural air as the anesthetist establishes complete lung expansion. It is important postoperatively to keep the lung completely expanded so that the graft is continuously pressed against its bed at all times.

CASE REPORTS

CASE 1.—M. L., an unmarried 24-year-old native Dutch West Indian woman, first came to Memorial Hospital on Nov. 9, 1945. She stated that in 1941, two weeks after a slight trauma to her left anterior chest well, she had noted a tumor mass in that area. It gradually increased in size and was intermittently painful. The following year it was excised, and the surgeon stated that it was a benign tumor originating in the pectoralis muscle. The mass soon recurred and was excised again in 1944. No report of the second operation could be obtained. Three months later the mass again recurred and the patient was finally referred to the United States for further treatment.

On admission physical examination revealed a well-developed, thin, Negro female in good general condition. The left anterior chest revealed a tumor mass measuring 5 x 5 cm. overlying the second and third ribs. This seemed to be attached firmly to the underlying structures. Below the mass there were two healed surgical scars, and beneath these scars there was another mass measuring 4 x 5 cm. Roentgenograms of the chest showed no evidence of metastasis to the lungs or destruction of ribs.

The patient was admitted to Memorial Hospital and on Dec. 14, 1945, from a biopsy of the tumor, the pathological report was spindle-cell sarcoma. On Jan. 5, 1946, an attempt was made to remove the growth. The subcutaneous tissue overlying the mass, the pectoralis major and minor muscles, and portions of the third and fourth ribs were removed with the tumor. The tumor was seen to invade pleura and no attempt was made to resect this pleural extension of disease.

Convalescence from the first surgical procedure was uneventful and the patient was readmitted to the hospital and operated upon on April 10, 1946. At this time, the left breast was raised as a flap from the chest wall, exposing the bony chest cage. The remaining tumor was excised, together with portions of the second, third, fourth, and fifth ribs, the intervening muscular bundles, and the underlying parietal pleura (Fig. 3), leaving a chest wall defect measuring 15 x 12 cm. A large graft of fascia lata was then removed from the right thigh and sutured into this defect. The lateral margin of it was sutured to the pleura and periosteum, and the medial margin was attached to the pleura, pericardium, and other

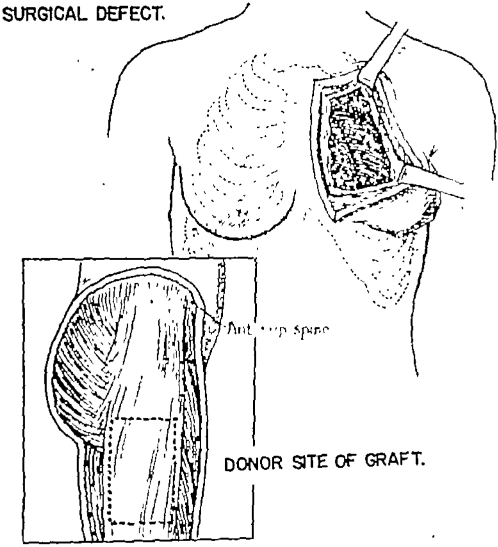

Fig. 1.—Surgical defect.

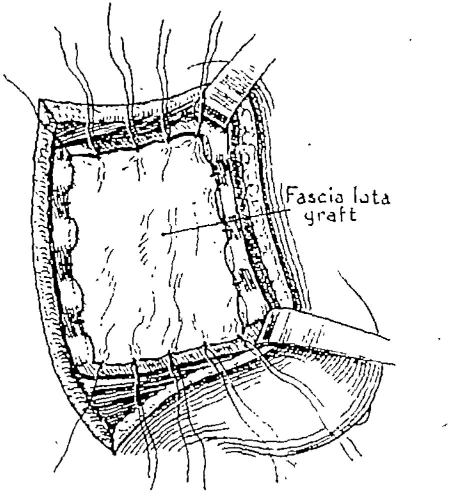

Fig. 2.—Free graft sutured in surgical defect.

soft tissues and muscles as they presented themselves. The skin flap and breast were then sutured in place over the fascia lata graft. Care was taken to fully inflate the lung before the operation was completed.

The patient tolerated the procedure very well, and the postoperative convalescence was without incident (Fig. 4). The lung remained well expanded, and there was no pleural effusion. Repeat postoperative x-rays showed no evidence of pleural reaction (Fig. 5).

The postoperative pathologic diagnosis was low-grade spindle-cell sarcoma.

CASE 2.—J. L., a 55-year-old white, unmarried female, entered Memorial Hospital Aug. 6, 1935, complaining of a lump in the left side of her abdomen which had been present, but symptomless, for two years. For several months prior to admission the mass had increased noticeably in size.

Physical examination was negative except for the tumor mass, which measured 12 x 5.5 cm. was elongated in shape, and seemed to originate near the left costal margin in the region of the anterior axillary line. The mass was elastic in consistency, firmly attached to the underlying bony structures, but not to the overlying skin. Roentgenograms of the chest showed considerable destruction of the left tenth and eleventh ribs.

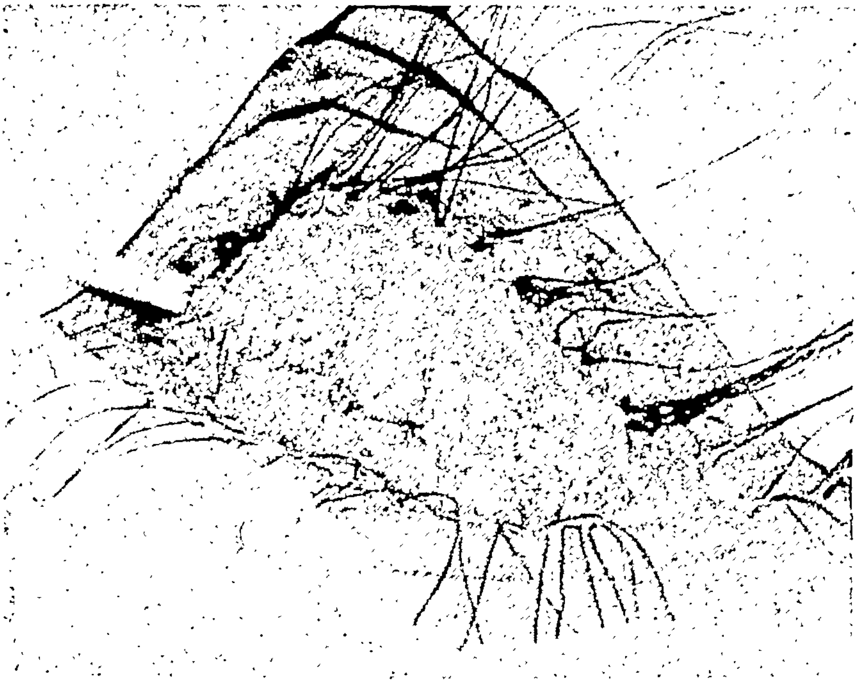

Fig. 3.—Graft in surgical defect.

The mass was aspirated and pathologic examination revealed it to be a chondromyxosarcoma. In preparation for surgery an artificial pneumothorax was established on the left side. On Aug. 16, 1935, operation was performed and a bulky tumor, measuring about 15 cm. in diameter was found to involve the anterior midportions of the left ninth and tenth ribs. It was adherent to the diaphragm and to peritoneum and could be palpated externally where it invaded the muscles of the anterior upper abdominal wall. Because of the adherence of the tumor, portions of the peritoneum, pleura, and diaphragm had to be excised, together with sections of the ninth, tenth, eleventh, and twelfth ribs (Fig. 6 *A* and 6 *B*). A long, wide strip of fascia lata was obtained from the left thigh and sutured to the diaphragm and pleura. The fascia lata graft actually substituted for a part of the diaphragm. The muscle tissue could not be approximated; the skin and subcutaneous tissues were sutured over the graft.

Postoperatively, the patient developed an effusion in the left pleural cavity and carried a few degrees of fever, but the wound healed well and sutures were removed by the eighth day. On the eighteenth postoperative day the patient suddenly went into shock and expired. An autopsy was obtained. The cause of death was found to be a thrombophlebitis of the left internal iliac vein with metastatic, bilateral, pulmonary emboli. Examination of the left pleural cavity showed an accumulation of a small amount of serosanguineous fluid. The graft had taken well to the diaphragm and pleura. There was no adherence of the graft to the visceral pleura.

The final pathologic diagnosis was low-grade chondrosarcoma.

CASE 3.—E. M., aged 21, entered the United States Naval Hospital, Brooklyn, N. Y., Oct. 23, 1944, because of a lump on the right chest. He stated that without known cause, a small mass had appeared on his right chest posteriorly and slowly increased in size, but caused him no pain or disability. An operation under local anesthesia at Quonset Point, R. I., Oct. 16, 1944, revealed the tumor, which was incompletely removed.

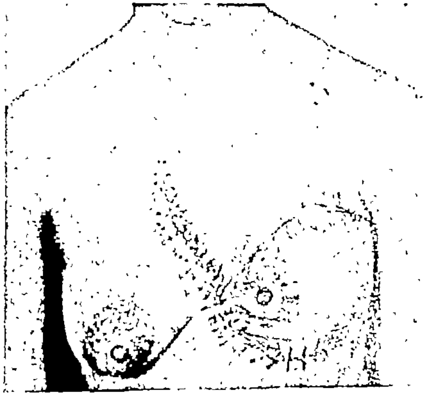

Fig. 4.—Postoperative appearance.

Physical examination revealed a recent scar 10 cm. in length over the right ninth rib posteriorly. The remainder of the physical examination was essentially negative.

On Nov. 29, 1944, a second operation was carried out and portions of the seventh, eighth, and ninth ribs resected on the right side with a block removal of the tumor and that portion of the chest wall. The resulting defect was closed with a broad sheet of fascia lata measuring 15 x 10 cm. 100,000 units of penicillin and 2 Gm. of sulfanilimide powder were placed in the wound. The tumor measured 18 x 16 cm., it was lobulated and movable except at its base, where it was seen to arise from the chest wall and ribs, and at its apex, where it *was intimately* adherent to the lung. The chest was closed without drainage. On Dec. 5, 1944, thoracentesis, right chest, yielded 700 c.c. Burgundy-colored fluid. On each of the following two days he was given 500 c.c. of whole blood. On Dec. 7, 1944, 1,050 c.c. of clear Burgundy-colored fluid was removed from the right pleural cavity. On Dec. 16, 1944, an x-ray showed the presence of a slight pneumothorax. Repeat x-rays of the chest showed a gradual disappearance of the pneumothorax and pleural effusion.

The postoperative pathologic *diagnosis was endothelioma of pleura.*

CASE 4.—R. G., aged 18, was admitted to the United States Naval Hospital, Brooklyn, N. Y., Sept. 30, 1943. He stated that during a routine physical examination in March, 1943, a symptomless mass involving the left fourth rib was discovered. The tumor was removed at another Naval Hospital in May, 1943, and a diagnosis of chondroma was made. In September, 1943, he began suffering from episodes of hemoptysis and at this time he entered the Brooklyn Naval Hospital.

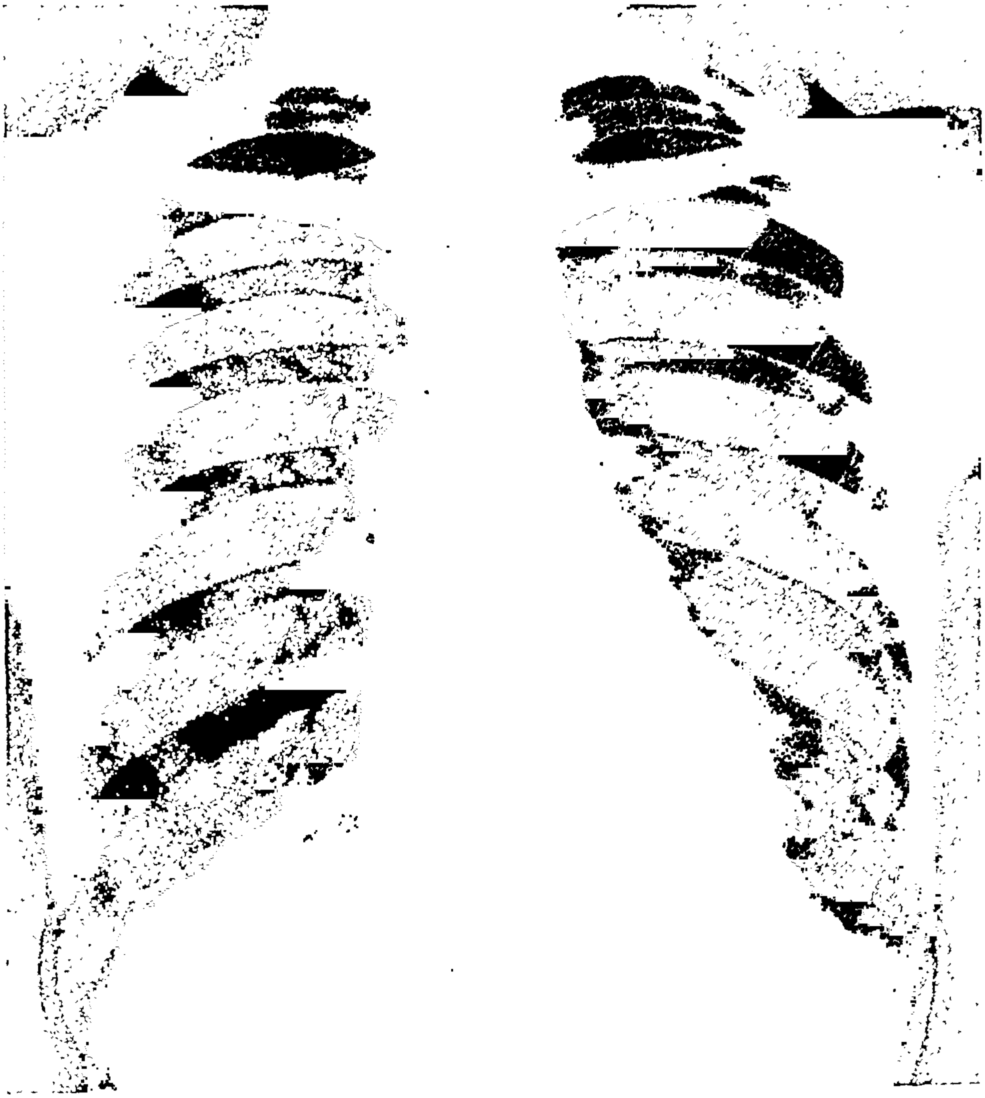

Fig. 5.—Roentgenogram eight days postoperatively.

Physical examination was essentially negative. There was a 4-inch, well-healed scar in the anterolateral aspect of the left chest in the area of the fourth interspace. There was no local evidence of recurrence. X-ray examination of the left thoracic cage showed evidence of a chondroma involving the partially-amputated anterior end of the fourth rib. After partial pneumothorax it was demonstrated that there was no fixation of the lung to the recurrent growth.

The patient was prepared for surgery and on Dec. 18, 1943, under cyclopropane, endotracheal anesthesia, a bony tumor of the left fourth rib together with the adjacent third rib was removed. This excision left a chest wall, pleural, and soft part defect which measured 8 x 10 cm. A fascia lata graft was next obtained and sutured into this defect.

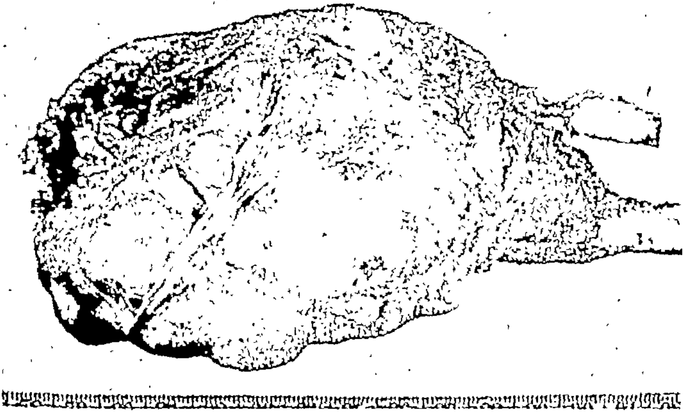

Fig. 6A.—Surgical specimen.

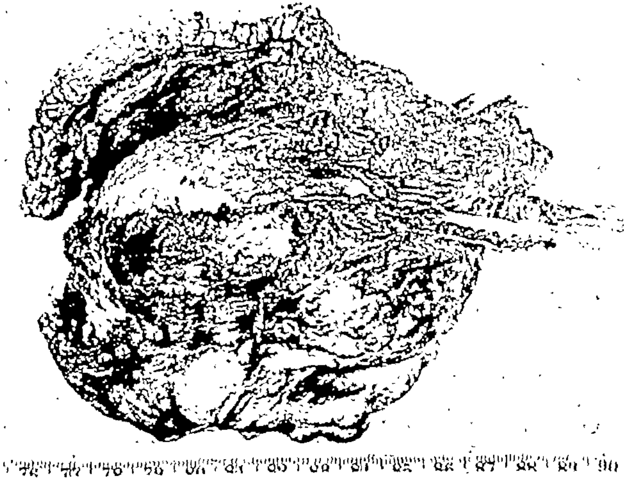

Fig. 6B.—Surgical specimen.

The postoperative course was essentially uneventful. There was some pleural reaction on the left side which subsided completely, and the incision healed per primum.

The final pathologic diagnosis was osteochondroma.

DISCUSSION

The use of fascia lata grafts in chest wall defects has several definite advantages:

1. It eliminates the need for a fixed, foreign body in the constantly expanding and contracting chest wall.
2. Fascia lata is readily available, even though it may not have been planned preoperatively to use a graft. If some other type of material were to be used, such as tantalum, definite preoperative plans would have to be made.
3. A better "cancer operation" can be carried out if one plans to help close the postoperative defect with a graft. A large graft of fascia lata will take as well as a small one, and one should not hesitate to resect at least one rib beyond the palpable extent of the tumor.
4. In cases of resection of the entire thickness of the chest wall, with exception of the skin, fascia lata adds a certain amount of support and also aids in making the chest wall airtight at time of operation.
5. Fascia lata grafts are firm enough to prevent postoperative pulmonary herniation.

The blood supply to the fascia lata grafts evidently comes through the side away from the pleural cavity. In Cases 2 and 3, the grafts took well in spite of varying amounts of pleural effusion. In the only case in which a postmortem examination was carried out, the fascia lata graft had taken well in spite of the presence of pleural fluid. Although, in these four cases, the procedure was used in the surgical care of primary tumors of the chest wall, it should also be applicable in cases of cancer of the breast with direct extension of disease into chest wall but without distant metastases.

SUMMARY

Four young patients had their large chest wall tumors resected and the resultant surgical defects closed with the aid of fascia lata grafts. The grafts took well in each case, and adequate chest wall support resulted in each instance. There was no evidence of postoperative herniation in any of the four cases.

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PUNCTURE WOUNDS OF THE CHEST

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PATIENTS with chest wounds are usually cared for by surgeons, and most of the literature on this subject is surgical. Unfortunately, this literature gives the impression that most of the care of patients with puncture wounds of the chest is surgical. Actually, only a very small proportion of the patients receiving such wounds have, in our experience, required surgery, if simple suture of a sucking wound can be called a prerogative of any doctor, or, if need be, of any medical corpsman. These wounds stand in sharp contrast to those of the abdomen, for instance, which entail painstaking surgery.

This report concerns the treatment on the medical service of a naval advanced base hospital of 100 consecutive patients with puncture wounds of the chest uncomplicated by severe wounds in other parts of the body. The patients were admitted some days after they had been wounded (Table I). Very commendable records, covering the period from wounding to admission, were received with the patients and pertinent data are drawn from them and from our own observations for use in the study. The patients were unselected, except that those with recognized empyema, a small proportion of the total, were usually sent directly to the surgical service.

TABLE I

	NUMBER OF DAYS ELAPSED BETWEEN DATE OF WOUND AND ADMISSION TO HOSPITAL																				
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	70
Number of patients (Total number of patients, 100)	0	1	8	12	12	12	6	7	5	6	6	8	4	2	2	3	0	3	1	1	1

In all cases, metallic bodies or fragments, or bone splinters, punctured four structures: the chest wall, one or more blood vessels, parietal and visceral pleuras, and lung. There was always some degree of pulmonary damage, usually hemorrhage into the pleural space, sometimes pneumothorax, and occasionally infection which subsequently led to empyema.

In forty of the patients a single bullet caused the wound. Thirty of these were accompanied by a wound of exit from the chest. In thirty-one the wound was caused by a single metal fragment, from shrapnel or bomb, and six of these had a wound of exit. In nine patients two or more fragments punctured the chest, but none of these produced wounds of exit. In twenty instances the cause of the wound was not known.

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Sucking wounds had been sutured before the patients arrived. Thirty-eight wounds were known to have been sutured, but how often suturing was required for physiologic and bacterial reasons and how often for cosmetic reasons is not known. Only two of the sutured wounds broke down sufficiently to allow communication with the chest cavity. Both appeared to result from empyema, rather than to cause empyema.

In all instances the period of shock had passed, and in nearly every instance the coughing of blood and of blood-stained sputum had ceased. A few patients were still distinctly dyspneic, but even in these, cyanosis was not conspicuous.

The physical findings varied widely, but usually included a healed wound of entrance, a healing wound of exit, the signs of fluid, and sometimes the signs of pneumothorax or consolidation. The roentgenologic examination of thirty-one of the chests showed the following: thirteen patients with free fluid only, the pulmonary collapse proportional to the amount of fluid; five patients with fluid and intrapulmonary metallic fragment; two patients with fluid and considerable air; one with fluid and atelectasis; one with fluid and consolidation; one with localized fluid only; one with "a slight pleural reaction"; one with a metallic fragment and a wedge-shaped area probably representing pulmonary hemorrhage; one with a single metallic fragment; and two with several metallic fragments, all without other signs; one with atelectasis only; and one entirely negative.

Roentgenologic examination of the chest was discontinued as a routine because x-ray usually failed to reveal important information not obtainable by physical examination. Its importance at a later stage is not denied.

Thoracentesis, other therapy, and the passage of time, usually lessened physical and roentgenologic findings.

Treatment was conservative. Good nursing care was stressed. Penicillin in doses of 20,000 units every four hours was usually continued for two to three weeks from the time of wounding, but this varied somewhat depending upon how "toxic" the patient was on admission. Sulfadiazine, either alone or in combination with penicillin, was used in some instances but for shorter periods than penicillin. Blood and plasma were restored as required. Oxygen was only occasionally necessary, though many of the men had required it in the first several days after being wounded (Table III).

Hemothorax and pneumothorax were reduced before admission in some cases but more commonly during the week after admission. At each aspiration of fluid one liter was withdrawn if it could be obtained and if distress by cough or pain did not interfere. Actually, the average successful (10 c.c. or above) aspiration yielded 622 c.c. If fluid was present but could not be aspirated, further trials of aspiration were delayed for one or more weeks and were then frequently successful. Some fibrin collections that at first prevented aspiration appeared later to autolyze. Sixty-seven of the 100 patients had one or more successful chest aspirations, counting those performed before the

TABLE II. HOSPITALIZATION AT BASE HOSPITAL

NO. DAYS	NO. PATIENTS
1- 5	0
6-10	27
11-15	17*
16-20	28
21-25	10
26-30	9*
31-35	1
36-40	1*
41-45	2*
46-50	1
51-55	2†
56-60	1†
61-65	1*
Total no. patients	100

*One of these patients had empyema.

†One of these patients was held in hospital for observation for a prolonged period, solely for study.

TABLE III. THERAPEUTIC-PROCEDURES (OXYGEN, PENICILLIN, SULFATHIAZOLE) BETWEEN TIME OF WOUNDING AND DISCHARGE FROM THE ADVANCED BASE HOSPITAL

NO. DAYS OF THERAPY	OXYGEN (NO. PATIENTS)	PENICILLIN (NO. PATIENTS)	SULFATHIAZOLE (OR SULFADIAZINE) (NO. PATIENTS)
1- 2	6	0	13
3- 5	2	5	10
6-10	2	31	19
11-15	2	23	4
16-20	0	23	5
21-25	0	9	0
26-30	0	2	0
31-35	0	2	0
36-40	0	0	0
41-45	0	1	0
Therapy given but number of days unknown	12	0	0
No therapy given	50	2	42
Total data available	80	98	93
Data not available	20	2	7
Total number patients	100	100	100

TABLE IV. THERAPEUTIC PROCEDURES (THORACENTESIS, TRANSFUSION, PLASMA) BETWEEN TIME OF WOUNDING AND DISCHARGE FROM THE ADVANCED BASE HOSPITAL

NO. PROCEDURES PER PATIENT	THORACENTESIS (NO. PATIENTS)	BLOOD TRANSFUSION (NO. PATIENTS)	PLASMA UNIT (NO. PATIENTS)
1	23	24	16
2	18	11	20
3	11	10	2
4	6	4	2
5	4	5	1
6	2	1	0
7	0	1	0
8	3	0	0
None given	30	35	50
Data available	97	91	91
Data not available	3	9	9
Total no. patients	100	100	100

patients were admitted (Table IV). In one instance moderate secondary hemorrhage occurred after thoracentesis. In none did infection seem to result from thoracentesis.

Much of the blood and plasma used was administered before the patients were received, but some patients required one or both after admission (Table IV).

Metallic foreign bodies and bone splinters deep in the wounds were not removed. Those near the surface had been removed in some cases before the patients were admitted, and in some cases were removed afterward. Fibrin collections were removed surgically in only one instance (a patient with empyema) at the time of rib resection and initiation of open drainage. None of the lungs were "decorticated."

Fever of considerable degree was recorded in many patients who proved to have no empyema. We believe the fevers were usually the result of absorption of protein breakdown products of the hemothoraces, even though there was no correlation between the extent of the hemothorax and of the fever. In forty-five patients who developed no empyema the oral temperature was 101° F. or above, seven days or more after the patients were wounded. In twenty-five of these the peak of fever, seven days or more after the patients were wounded, was 102° to 102.8° F. In twelve others the peak was 103° to 105° F.

Sustained fevers above 103° F. strongly suggested the onset of empyema, and those patients required frequent diagnostic aspirations and culture of the fluid. When the fluid lost its thin appearance, contained shreds, had a foul odor, contained many pus cells in smear, and yielded bacteria on culture, there was no doubt that empyema had developed, and the patient was transferred to the surgical service for rib resection and drainage. The five patients in this category had peaks of fevers of 102.5°, 103.0°, 103.0°, 103.8° and 105.0° F. respectively, and all had empyemas. Four of the five empyemas yielded pure culture of *Staphylococcus albus* or *aureus*. When fever was moderate and the patient was not toxic, a single positive culture was not considered diagnostic of empyema because several patients with positive culture in the absence of confirmatory findings failed to develop empyema. Penicillin administered systemically appeared to have some bacteriostatic action in cases of suspected empyema that failed to materialize. On the other hand, once empyema was established, penicillin administered intramuscularly (20,000 units every fourth hour) and intrathoracically (100,000 units daily) had little effect. In one instance, intrathoracic penicillin appeared to lower the fever somewhat, but the daily drainage of fluid may have contributed to the effect.

Cardiac involvement was suspected clinically in seven cases. None of these patients required a pericardial tap and none evidenced pericardial sepsis. One of two who developed pericardial friction rubs had faint heart sounds and a cardiac silhouette by roentgen examination suggestive of fluid. Their electrocardiograms showed T-wave flattening or shallow inversion in various leads (Figs. 1 and 2).

Three patients, twelve to twenty-one days after being wounded, developed precordial or substernal pain with left shoulder radiation associated with dyspnea and signs of shock. Their tracings showed more marked abnormalities, consisting in two instances of biphasic T-waves in Lead I and sharply inverted T-waves in Lead IV-F (Figs. 3 and 4). The third patient had inverted T-waves in the limb leads with slight RS-T segment elevation in Leads II and III (Fig. 5, *A*). A tracing five days later showed deeper inversion in these latter leads with disappearance of the RS-T segment displacement (Fig. 5, *B*). We are unable to offer an explanation for the relatively late development of cardiac symptoms in these three cases.

The remaining two patients had no cardiac symptoms or clinical signs but the location of their wounds led us to suspect cardiac injury. The electrocardiograms of one showed T-wave inversions in Leads I, II and IV-F which became more marked after five days (Fig. 6, *A* and *B*). The other patient had a deep Q-wave in Lead I, smaller Q-waves in Leads II and IV-F and T-wave abnormalities most marked in Lead I (Fig. 7).

Electrocardiograms were taken on fifteen patients with no clinical evidence of cardiac or pericardial involvement. Nine had left, and six had right hemothoraces. Seven of the patients having left hemothoraces had normal tracings and two had transient diminution of T-wave amplitude. These latter had a mediastinal shift but so did three of the patients whose electrocardiograms were normal. Of the six patients with right hemothorax, three had right axis deviation and one had a vertical axis.

Thus electrocardiography consistently showed changes compatible with pericardial or myocardial abnormality in the 7 per cent of our patients having clinical signs or symptoms referable to the heart. Associated hemothorax and mediastinal shift did not produce misleading electrocardiographic changes.

The average duration of hospitalization of all of the hundred patients with chest wounds was nineteen days (see Table II). All but two of the patients appeared to improve. One of these two died. Most of the patients improved conspicuously. Four of those with empyema were free of fever when evacuated.

Evacuation was accomplished by air transport in most instances, but some was by hospital ship. Evacuation by ships not primarily designed for hospital work was avoided because patients with chest wounds might develop needs for surgery. Patients with pneumothorax were evacuated by hospital ship, and were not exposed to risk of expanding pneumothorax at increased altitudes.

The one patient who died was wounded fifteen days before admission to the advanced base hospital. A bullet had entered the third interspace at the left sternal margin and emerged from the left anterior axilla and had produced a sucking wound which had been sutured at an advanced field hospital. He denied hemoptysis. He was at first extremely dyspneic. One liter of bloody fluid was aspirated from the left chest. Eight days after he was wounded, x-ray examination showed almost complete collapse of the left lung, with a fluid level at the ninth rib posteriorly. At that time the temperature and pulse were only slightly elevated, and respirations were not severely difficult.

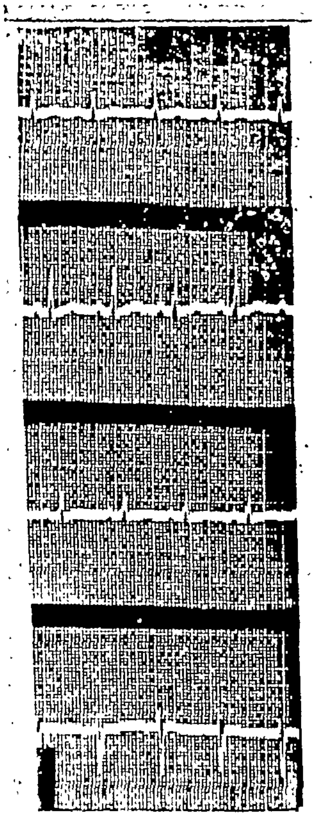

Fig. 1.

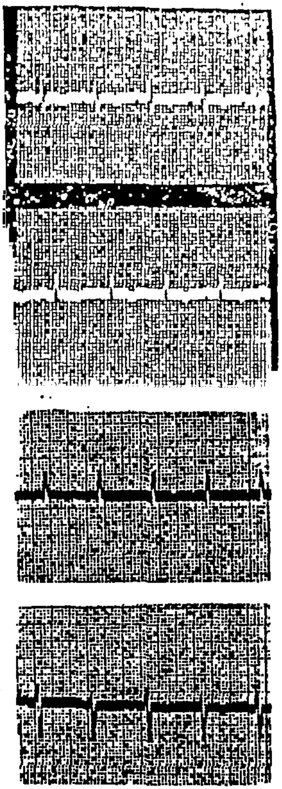

Fig. 2.

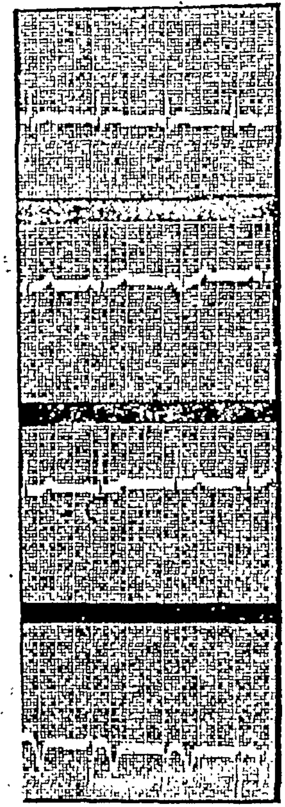

Fig. 3.

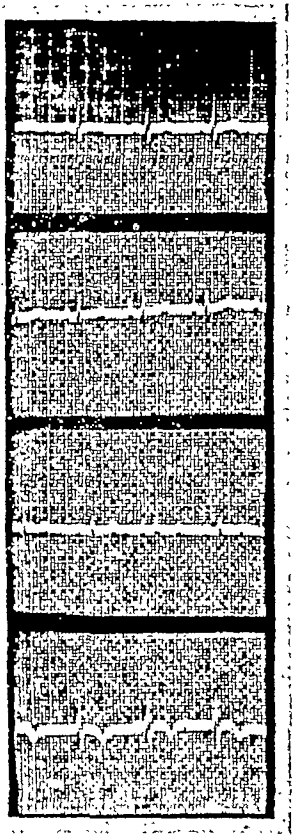

Fig. 4.

Electrocardiograms taken on patients having cardiac injury as a complication of their chest wounds. For description, see text.

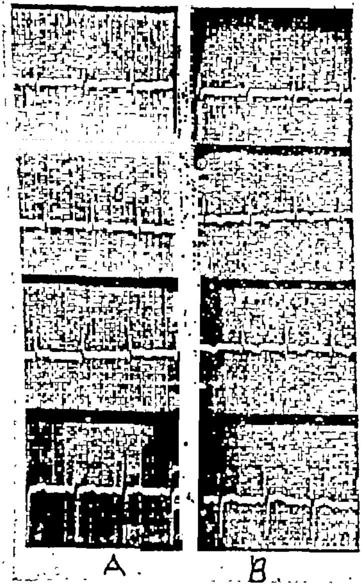

Fig. 5.

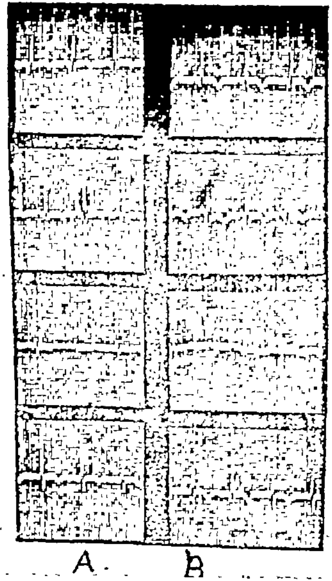

Fig. 6.

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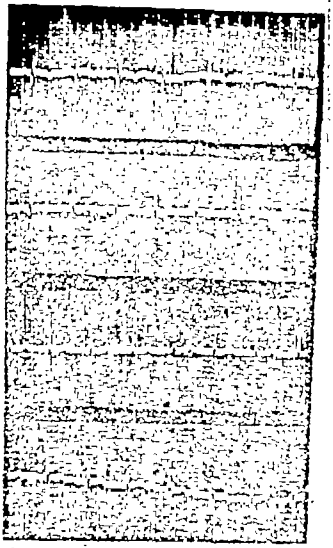

Fig. 7.

During the following ten days, in spite of penicillin therapy, he had one, then two, and finally four and one-half degrees of fever. Only one cubic centimeter of a cloudy fluid was recovered by thoracentesis. He was transferred to the surgical service. Thoracotomy was performed, and drainage was established. The toxic state failed to diminish. Further exploration was unsuccessful. He died several days later. Autopsy showed a single, drained, left empyema cavity with no encapsulated pus, little or no expansion of the lung, some hemorrhage into the pleural cavity, and toxic hepatitis and splenitis.

DISCUSSION

Most patients with chest wounds resulting from this war have been handled in much the same way as were those in the present report.[1] Our cases are not presented because of originality. They are presented in order that one can understand what to expect of one phase of conservative therapy. Unfortunately, there is no opportunity at the present time to follow our patients through to complete recovery.

The treatment of chest wounds we have employed can best be called the conservative method. In all but the five instances in which the patients developed empyema, it was carried out on a medical service without benefit of surgery. In the case of the patients with empyema, usual rib resection and open drainage were performed by the surgeons.

Considering the shocking nature of the wounds, the patients improved very satisfactorily, and most were on their way to complete recovery when discharged from the hospital. In some instances in which fibrin collections prevented aspiration and interfered with re-expansion of the lung, the fibrin later autolyzed and expansion was accomplished by successful aspiration. In some instances fibrin collections did not autolyze. Empyemas were remarkably exceptional.

Other less conservative methods of treating puncture wounds of the chest have been used. Much has been written during the war years of surgical intervention to remove fibrin collections.[2 and 3] Its advocates suggest the third to tenth week as the optimum time for this, since some believe that irreversible encapsulation of incompletely expanded lung may result from fibrin collections within that time. Other surgeons, some of the British for example, believe major surgery should be utilized on the day of wounding, after the patient has recovered from shock.[4] They advocate exploration of the chest under closed anesthesia, suture of the bleeding vessels, evacuation of all blood and clot, rinsing of the pleural space with physiological saline, and maintenance of artificial pneumothorax. Most American surgeons insist that pneumothorax should be avoided, or reduced as promptly as possible.

Good results have been obtained by these surgical methods when carried out by competent thoracic surgeons. We doubt that they afford lowered mortality or shortened morbidity. Their practicality, especially at advanced bases in average hands, is not proved, and statistics of end results are incomplete.

Statistics concerning the relative validity of various methods of treating wounds of the chest could be obtained from the records of the medical corps of the services and from a follow-up of wounded to the time of recovery. This would help immeasurably in choosing the best methods of treating chest wounds.

SUMMARY

The conservative medical management of 100 patients with puncture wounds of the chest at an advanced naval base hospital has been outlined. All but two of the patients improved, and all but one lived. Five developed empyema and four responded well to surgical drainage. We hope this will stress the need for a statistical comparison of the end results of treatment by conservative methods and by surgical intervention.

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SODIUM TETRADECYL SULFATE, AZOCHLORAMIDE, AND SULFANILAMIDE MIXTURE IN THE PREVENTION OF EMPYEMA FOLLOWING PNEUMONECTOMY WITH CONTAMINATION

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ENCOURAGING results in attempts to sterilize the pleural cavity in pyogenic empyema with instillations of an antiseptic solution[3, 10] led the authors to extend its use to the prevention of empyema following pneumonectomy.

Contamination of the pleural cavity during pneumonectomy in cases of lung abscess, bronchiectasis, or carcinoma complicated by lung abscess is a great hazard in thoracic surgery. The empyema which usually follows such contamination presents a serious problem in treatment. Patients afflicted with such a condition continue to drain for months, or death from sepsis and debilitation may occur. Following prolonged drainage of the pleural cavity, obliteration by thoracoplasty offers the best chance for recovery. Complete obliteration can only be accomplished by the multiple stages of a thoracoplasty. This is a costly, time-consuming, and sometimes hazardous procedure, especially for patients who already have been ill, perhaps for many months, and who have been subjected to pneumonectomy.

The search for a solvent for digestion of sputum and empyema fluid in the concentration examination for tubercle bacilli[5, 6] led to the use of detergents or wetting agents. Of the numerous detergents used in experiments, sodium tetradecyl sulfate was the most satisfactory. It had been known for many years that soaps enhanced the effect of bactericidal agents. The wetting agents which possess soap properties in a greatly intensified degree were then selected as a basis for further study in combination with antibacterial solutions.

The properties of the wetting agents explain their effectiveness for this purpose. Because of the dual nature of the structure of their molecules wetting agents have a tendency to locate themselves on an aqueous surface or interface, thus forming a monomolecular film. For this reason, the surface tension of solutions coming in contact with the agent is reduced. Highly surface active solutions can, therefore, penetrate into openings and cracks, crypts and recesses, which a conventional aqueous saline solution would simply bridge. Solutions of wetting agents can even wedge their way into the center of clumps of bacteria, leucocytes, and debris.

With these ideas in mind Petroff and Schain[4] experimented with the bactericidal properties of various antiseptics reinforced with different detergents. Of the many combinations of compounds used, azochloramide and sodium tetradecyl sulfate solution were found to be the most effective. Animal experiments

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as well as clinical observations have shown that sodium tetradecyl sulfate is well tolerated by mammals when administered orally, intravenously, or when injected into the pleural cavity.[1, 2]

Following the experimental work of Petroff and Schain, Palitz and Herman[7] described the treatment of tuberculous and mixed infection empyema with azochloramide-sodium tetradecyl sulfate solution. Successful results were noted in 81.5 per cent of the cases without bronchopleural fistulas. In general, following the irrigations they reported that the fever was lowered and the fluid was thinned. Sinuses and abscesses were affected favorably.

Brantigan and Owings[3] followed up this work and reported the use of sodium tetradecyl sulfate and azochloramide in the treatment of acute pyogenic empyema of the pleural cavity. Two patients were treated and the results proved outstanding.

In 1941, Neter[4] reported on the combined bacteriostatic activity of sulfanilamide and azochloramide upon Group A hemolytic streptococcus and enterococcus. His experiments revealed that sulfanilamide (1,000 mg. per cent) used in combination with azochloramide (1:800,000) may prevent completely the growth of enterococcus and exert greater bacteriostatic activity than azochloramide alone, even in a concentration twice as great.

Schmelkes and Wyss[9] corroborated this work. Their experiments indicated that the bactericidal effect of sulfanilamide in the test tube can be enhanced greatly by small concentrations of azochloramide which are ineffective in themselves. Similar concentrations of azochloramide will also restore the bactericidal effect of sulfanilamide in the presence of para-aminobenzoic acid (which neutralizes the action of sulfanilamide), suggesting that the enhancement observed in the first place is attributable to the inactivation of sulfanilamide inhibitors by the azochloramide.

The results of the work of these men indicate that the combination of sodium tetradecyl sulfate, azochloramide, and sulfanilamide is a highly effective nonspecific bactericidal agent. The authors have placed it in the pleural cavity at the termination of all open chest operations since 1943. In uncontaminated thoracic operations it seems to have eliminated all empyema. However, there is no proof that infection would have occurred without its use.[1, 2] The mixture seems also to reduce pleural exudation, which, of course, reduces or eliminates the necessity for postoperative pleural aspirations following open thoracic operations.

Since adopting the routine use of the mixture in the pleural cavity after open chest operations, four cases of gross contamination of the pleural cavity occurred during pneumonectomy. The results in these four cases were so highly satisfactory that they are being reported.

CASE REPORTS

CASE 1.—W. S. was a white man aged 32. In 1942 a carcinoma of the testicle was removed. A submucous nasal resection was performed in March, 1944. In October of the same year a diagnosis of disease in the right lung was made; malignant metastasis from the testicle was suspected. The disease in the right lung became more extensive, and the patient, weaker. The left lung was found to be normal. Based upon the possibility that malignancy might not be the cause of his condition, operation was advised and accepted. By this time the presence of multiple lung abscess cavities was definite. On May 2, 1945, under general endotracheal anesthesia, a right pneumonectomy was performed. During operation one lung abscess cavity was entered and foul-smelling pus was spilled into the pleural cavity. Sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 in 200 c.c. quantity and 5 Gm. of sulfanilamide were placed in the pleural cavity. The chest was closed without drainage. The patient's course was uneventful and his temperature normal until the third postoperative day, when he began to have slight fever. Empyema of the right pleural cavity could not be diagnosed until the eighth postoperative day.

After the diagnosis of empyema, aspirations and instillation of the sodium tetradecyl sulfate, azochloramide and sulfanilamide mixture would eliminate his symptoms but would not cure the infection. A pure culture of *Staphylococcus aureus hemolyticus* persisted. Open drainage of the pleural cavity was done, followed by a multiple stage thoracoplasty approximately six months later. A satisfactory cure has resulted. There was no sign of malignancy in the lung that was removed.

Comment.—From a study of the patient it appeared evident that the single application of the antiseptic solution killed all bacteria except the *Staph. aureus hemolyticus,* inhibited the growth of these bacteria for three days, and definitely retarded their growth for several more days. The solution failed to kill all the bacteria and true empyema developed. It seemed logical to believe that perhaps too little solution had been used. The antiseptic, of course, was diluted by blood and serum oozing into the pleural cavity and perhaps even changed in chemical consistency. It was decided that when the next gross contamination of the pleural cavity occurred with pneumonectomy a larger amount of the solution would be used, followed by aspiration of the pleural cavity and instillation of the sodium tetradecyl sulfate, azochloramide and sulfanilamide mixture each day after operation until three such treatments had been given.

CASE 2.—J. C. was a colored man, aged 43. In June, 1945, his chief complaint was pain in the left side of the chest accompanied by fever for a few days. A roentgenogram made at this time revealed a lesion in the region of the hilus of the left lung, with ill-defined cavities. Sputum studies failed to show tubercle bacilli. Bronchoscopic examination then confirmed the impression of multiple lung abscesses. On admission to the hospital the patient was running a febrile course with daily elevations of temperature to 102° F. by mouth. Operation for pneumonectomy was advised and accepted by the patient. Therefore, on Nov. 11, 1945, under general endotracheal anesthesia, a left pneumonectomy was performed. During the operation the major bronchus of the left lung was accidentally opened, thus spilling foul-smelling pus into the pleural cavity. Prior to the closure of the chest without drainage approximately 500 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with 5 Gm. of sulfanilamide was placed in the pleural cavity. On the first, second, and third postoperative days the chest was aspirated and instilled with the same mixture. The patient's postoperative course was uneventful. His temperature was normal on the seventh postoperative day. The wound did not drain and the pleural cavity did not become infected. He was discharged Dec. 2, 1945, and has remained well. The pathologic diagnosis was multiple abscesses of the left lung.

CASE 3.—S. P. was a 46-year-old white man who was admitted to the hospital with diagnosis of abscess in the upper lobe of the left lung. The patient reported that his present illness began in December, 1945. Following admission to the hospital he ran a low-grade fever for three weeks. On his twenty-fifth hospital day he experienced an episode of sharp pain in the left side of the chest accompanied by increased temperature elevation, accelerated pulse rate, and bloody sputum. A physical examination showed evidence of thrombophlebitis of the left femoral vein. Therefore, on Aug. 15, 1946, a ligation of the common iliac vein on the left side was performed. It was postulated at this time that the patient's lung abscess may have been caused by a previous pulmonary infarct. It was decided to watch the patient further in the hope that his lesion might spontaneously improve following obliteration of the source of the emboli. He was observed for a two-month period, during which time he periodically ran a low-grade fever and the chest lesion seemed to increase in size.

Exploratory thoracotomy was advised and accepted by the patient. On Oct. 8, 1946, under general endotracheal anesthesia, the left pleural cavity was explored. An examination of the upper lobe of the left lung revealed a lesion that was thought to be a bronchogenic carcinoma complicated by a lung abscess. A left pneumonectomy was performed; however, during the course of the operation the left pleural cavity was grossly contaminated by several cubic centimeters of foul-smelling pus which was spilled when the abscess cavity in the lung was entered inadvertently. Prior to closing the chest without drainage 500 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1,3,300 with 5 Gm. of sulfanilamide was instilled into the pleural cavity.

The patient's temperature, which had been 101° F. several days prior to operation, was 100° F. on his first postoperative day. The chest was aspirated at this time and 400 c.c. of bloody fluid removed; 300 c.c. of sodium tetradecyl sulfate, azochloramide, and sulfanilamide mixture were instilled into the left pleural cavity. On the second postoperative day his temperature reached 102° F. and the same procedure was repeated. On the third postoperative day his temperature rose to 101° F. Thoracentesis was repeated and 350 c.c. of bloody fluid was aspirated; 300 c.c. of sodium tetradecyl sulfate, azochloramide and sulfanilamide was placed in the pleural cavity. On the fourth postoperative day the patient's temperature returned to normal, where it remained for the duration of his stay in the hospital. The wound did not become infected, nor did infection of the pleural cavity occur. The patient was discharged on his twenty-fifth postoperative day. The pathologic diagnosis was bronchiogenic carcinoma with bronchiectasis and abscess formation.

CASE 4.—J. M. was a 35-year-old white man who was admitted to the hospital with a diagnosis of bronchiectasis of the left lung. His past history was significant because in 1918 he had bilateral pneumonia followed by empyema of the left pleural cavity. An operation for open drainage was performed; the patient recovered and was asymptomatic until 1932, when he had hemoptysis. He continued to cough blood occasionally and in the last few years prior to admission hemoptysis had become more frequent. Bronchography revealed an extensive cystic type of bronchiectasis throughout the left lung from the apex to the base. The right lung was normal. The heart was markedly retracted to the left side.

On Oct. 18, 1946, under endotracheal anesthesia, a pneumonectomy of the left lung was performed. During the operation, which was accomplished with great difficulty, it was evident from the appearance in the chest that a bronchiectatic abscess cavity had been entered. This was confirmed by examination of the lung after completion of the operation. Prior to closure of the chest without drainage 1,000 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with 5 Gm. of sulfanilamide was placed in the pleural cavity. On the following day 700 c.c. of bloody fluid were aspirated and 200 c.c. of sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,300 with sulfanilamide were inserted into the pleural cavity. On the second postoperative day 450 c.c. of bloody fluid were aspirated and 200 c.c. of the sodium tetradecyl sulfate-azochloramide solution with sulfanilamide were instilled. On the third day the same treatment was repeated. During this period the patient ran a low-grade febrile course with occasional elevations of temperature to 101° F. by mouth. On the fifteenth postoperative day 450 c.c. of serosanguineous fluid were again aspirated. No instillation was done. The patient remained free of wound infection or empyema and was discharged from the hospital on the twenty-fifth postoperative day. The pathologic diagnosis was bronchiectasis of the left lung.

Comment.—All the patients, except in Case 1, received 30,000 units of penicillin by intramuscular injection every three hours. However, penicillin was not placed in the pleural cavity at the time of operation, nor was it instilled later into the pleural cavity in any of the four cases reported.

One important aspect is obviously deficient in this report. Cultures of the pleural cavity were not obtained at the time of operation. Nevertheless, in each case several cubic centimeters of pus were spilled into the pleural cavity. Subsequent cultures of the aspirated pleural fluid were absent when the charts were reviewed. The few culture reports available have been omitted purposely because they are incomplete and have a tendency to strengthen the beneficial effects noted clinically.

This report seems justified since the method of treatment has succeeded in preventing empyema in three consecutive patients where gross contamination of the pleural cavity occurred during pneumonectomy. These patients have been followed since their discharge from the hospital and all have remained well.

In Case 1 the solution would not cure an established empyema caused by hemolytic *Staph. aureus.* However, it is important to point out that a pleural space that cannot be obliterated, as one finds after pneumonectomy, presents an entirely different problem from empyema where there is a lung that can expand enough to finally obliterate the pleural space. Where a lung is present to obliterate the pleural space, even though a few bacteria survive the antiseptic they will be trapped and killed off by the body defenses. In an endeavor to sterilize chronic empyema cavities where obliteration of the pleural space could not be accomplished, the solution usually will quickly eliminate all bacteria except hemolytic *Staph. aureus* or *albus.* It will keep these inactive so long as the solution is changed at intervals of forty-eight hours.

In the three cases presented the treatment is directed at killing the contaminating bacteria before infection or empyema can become established. It seems logical that an attack upon any bacteria at this stage would be more likely to eliminate them than an attack made after they had successfully invaded the tissues. Of course, it is possible that hemolytic *Staph. aureus* or *albus* was not present in the pus contaminating the pleural cavity of the cases reported.

Since sulfanilamide is poorly soluble, it is impossible to determine how much of the drug enters the pleural cavity upon the instillation with syringe and needle. Usually 5 Gm. of sulfanilamide was placed in 500 c.c. of the solution of sodium tetradecyl sulfate 1:500 and azochloramids 1:3,300. The mixture is agitated and then drawn into the syringe and instilled; thus, only an indefinite amount is actually deposited in the pleural cavity. However, with the chest open at the operating table, the sulfanilamide is readily deposited as desired. The mixture should be prepared fresh prior to usage, since azochloramide is an unstable preparation. In aqueous solution this compound decomposes and gives up its chlorine; it therefore decreases in potency when left exposed to air and light. The mixture should be used at approximately body temperature. The sudden introduction of this solution into the pleural cavity may bring about temporary respiratory arrest in the anesthetized patient. The period of apnea is of short duration, but it is well for the anesthetist to realize that a period of apnea may occur.

CONCLUSIONS

Sodium tetradecyl sulfate, 1:500 in azochloramide, 1:3,000 with sulfanilamide is an efficient nonspecific bactericidal agent.

When the mixture was properly applied to the pleural cavity after pneumonectomy, it prevented empyema in three consecutive cases where gross contamination occurred during pneumonectomy.

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PULMONARY TUBERCULOSIS ASSOCIATED WITH ANOMALOUS COMMON LEFT PULMONARY VEIN ENTERING THE LEFT INNOMINATE VEIN

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THE perfection of technique for resection of the lung and its segments has called particular attention to the structural relationship about the roots of the lungs. This increased interest has been responsible for the introduction into the surgical literature in recent years, of various anomalies of the pulmonary circulation which have been encountered at operation and in the autopsy room.

Zuckerkandl,[1] in 1881, called attention to normal communications between the systemic and pulmonary circuits, and Brown[2] developed the present embryologic conceptions that explain the presence of wide variations from the normal in the termination of the pulmonary veins. Harris and Lewis[3] mention Brown's study of the development of the two vein systems from an undifferentiated celomic plexus, and state that in the adult, anomalies of the pulmonary veins will be of two types: in the upper chest there will be communications between the pulmonary veins and tributaries of the superior vena cava; and in the lower chest there will be those between the pulmonary veins and the hepatic portion of the inferior vena cava. The persistence of these embryologic communications is due, they believe, to inadequate development of the proximal portion of the cephalic tap (which later becomes the common pulmonary vein) resulting in the maintenance of the distal communications with the anterior or posterior cardinal vein or the caudal tap, the latter the precursor of the portal vein.

Maude Abbott[4] mentions the occurrence of anomalous pulmonary veins associated with cardiac defects and cites one case with a left pulmonary vein entering the left subclavian and the right pulmonary vein entering the portal vein. She states that wide variation from the normal may be associated with few symptoms. Brody[5] was able to collect 100 cases from the literature and added two of his own in which abnormal pulmonary veins were recorded. Many of them were associated with heart defects. He stated that, if 50 per cent of the pulmonary blood reached the left side of the heart, an adequate oxygen level would be sustained. Of fifteen such cases, he notes one, which was reported by Dean and Fox,[6] of a person who lived 86 years. This man had a common left pulmonary vein entering the left innominate vein. Compere and Forsyth[7] believe that if 75 per cent of the pulmonary blood reaches the left side of the

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heart, there will be no recognizable cardiorespiratory symptoms. Their case of an anomalous right upper lobe vein which entered the superior vena cava showed no circulatory difficulty during a fifteen-year period of observation for central nervous system syphilis.

This report concerns an anomalous common left pulmonary vein entering the left innominate in a patient with pulmonary tuberculosis under observation for over three years.

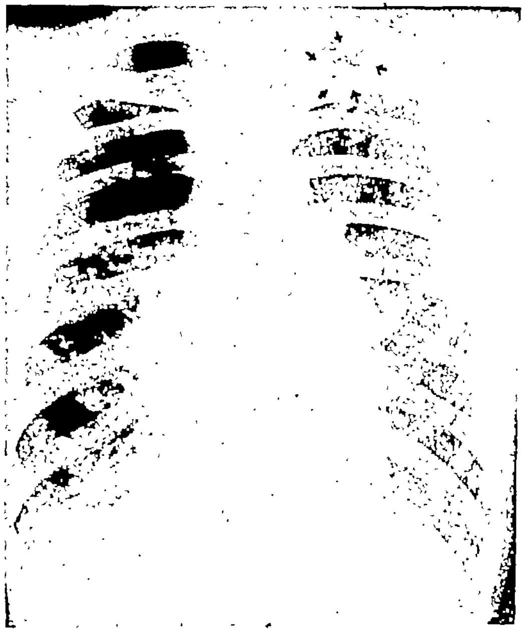

Fig. 1.

CASE REPORT

J. C., aged 37, was a Negro male, unskilled laborer. Bilateral pulmonary tuberculosis was discovered Nov. 27, 1942, when the patient was examined by x-ray prior to a hemorrhoidectomy at Freedmens' Hospital, Washington, D. C. (Fig. 1). The x-ray film showed fine nodular infiltration extending outward from the root into the middle third of the right lung field. The left lung showed scattered nodular infiltration above the third rib, anteriorly, with a 3 cm. cavity at the apex.

The lesion on the right side gradually improved but the left apical cavity increased in size until it extended down to the second rib, anteriorly. Left pneumothorax was induced in May, 1944. This was complicated by extensive, inoperable, apical adhesions and the pneumothorax was abandoned. In January, 1945, the left apical cavity had increased to 5 x 10 in size, and the lesion in the right lung had nearly completely resolved. In January, 1946, the left apical cavity occupied the entire apical region above the third rib, and the left lung began to show reactivation between the second and fifth ribs. During this period the man was a patient on the tuberculosis service of Freedmens' Hospital, but in March, 1946, it was necessary to discharge him as a disciplinary problem. He worked as a bootblack for about a month and then sought hospitalization when acute symptoms developed. In reviewing the case, Freedmens' Hospital noted no respiratory embarrassment during the entire stay at that institution.

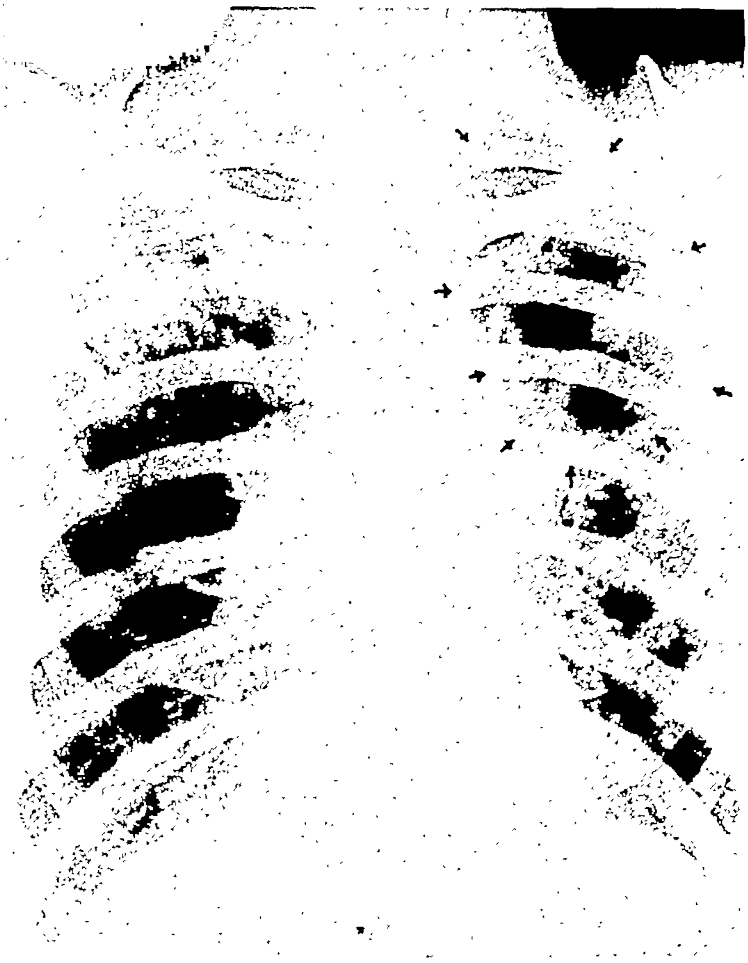

Fig. 2.

On admission to Glenn Dale Sanatorium May 3, 1946, the patient was critically ill, febrile, toxic, and dyspneic. The blood pressure was 136/88. There was no clubbing of the fingers and no evidence of cardiac disease (Fig. 2). The x-ray showed soft, mottled infiltration above the third rib, anteriorly, on the right, with the presence of several small cavities. The left lung contained a giant cavity above the third rib, anteriorly, with fine scattered infiltration to the base. His condition did not improve on bed rest, he developed a right pleural effusion, and died July 17, 1946.

Autopsy was performed July 20, 1946. The right lung was found densely adherent to the chest wall except at the base, where there was a pocket of clear pleural fluid about one liter in amount. Injection and tubercle formation were present over both visceral and parietal pleurae in this region. On section the lung showed marked consolidation throughout with several small cavities in the upper lobe.

The left lung was densely adherent to the chest wall and showed a 10 cm. apical cavity in the upper lobe and much caseation throughout the remainder of the lung parenchyma.

The heart was slightly enlarged, showing moderate dilatation of the right side and scattered small areas of atheromatous change in the aorta. There were no defects in the interauricular or interventricular septa. The left auricle received only one pulmonary vein, that from the right side, which divided as it passed through the pericardium. The circulation of the right lung was normal. Further exploration of the pulmonary circulation showed a common left pulmonary vein emptying into the left innominate and picking up a tributary from what appeared to be the left hemiazygous above the level of the left pulmonary artery (Fig. 3). Except for its origin in the interlobular fissure in the cardiac sulcus of the lower lobe, the anomalous vein was entirely superficial, running anterior to the pulmonary artery, then proceeding along the mediastinum to its termination. The vein to the upper lobe was exceedingly short. One interlobar vein was found.

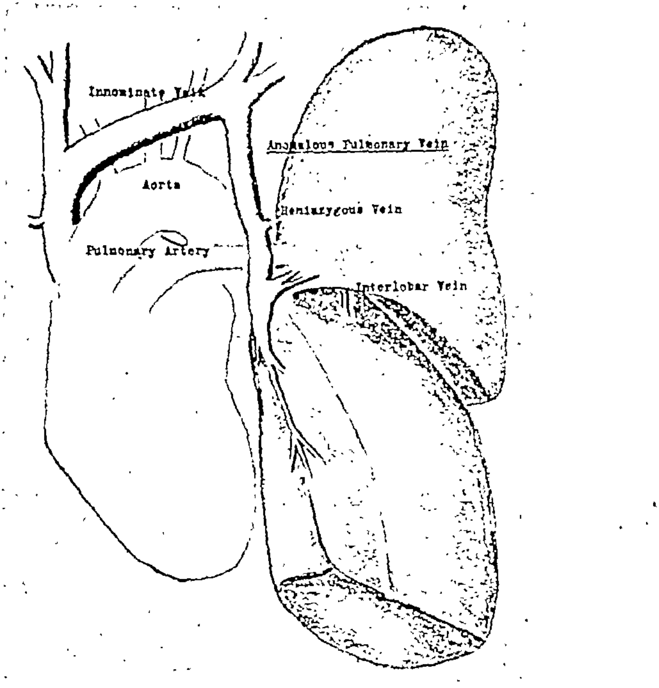

FIG. 3.

Extrapleural dissection was required to expose the left lung intact, and it is possible that communications with other veins of the chest wall may have been destroyed in the process.

COMMENT

It would appear that by itself recirculation of aerated blood from one lung into another, by a shunt involving half the pulmonary bed, still provides an adequate pulmonary reserve. In the case reported, the arterial blood received all its oxygen from the right lung. The addition of pulmonary disease to the picture suggests in this instance that the integrity of the right lung was more important than the left. Obstruction to the flow of blood through the right lung, which was directly concerned with maintaining the oxygenation of the systemic circuit, had a more serious effect than involvement of the left lung. Up to the time the patient suffered his terminal episode, the tuberculosis was more extensive on the left side. When spread to the right lung occurred and was associated with a right-sided effusion, the supply of oxygen and blood to the systemic circuit then became limited.

The surgical implications of this anomaly are apparent. Because of the density of the mediastinal adherence about the left root, superficially, this anomaly had the appearance of the left superior vena cava reported by Lam.[8]

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ANOMALY OF AORTA SIMULATING MEDIASTINAL TUMOR

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Solitary tumors of the mediastinum tax the diagnostic acumen of the clinical surgeon. Symptoms referable to the tumor may be either absent or of little importance. A careful history and physical examination usually fails to disclose the presence of a tumor in the mediastinum.

Roentgenologic examination is our most important method of diagnosis. It frequently directs attention to unsuspected mediastinal pathology. In addition, it offers valuable information pertaining to the type of tumor by localizing the mass, by revealing its relationship to adjacent structures, by depicting its configuration, and by establishing the presence or absence of pulsation. Diagnostic roentgen therapy also plays a role in the differential diagnosis in certain instances, by determining radiosensitivity.

All too frequently the preoperative studies fail to establish with any degree of accuracy the type of tumor present. In a group of 114 patients operated upon for mediastinal tumors at a number of Army Thoracic Surgery Centers during the last war,[1] five were found to have unsuspected aneurysms, and in five cases benign inflammatory lesions were encountered. One of us, F. P. C., in an unpublished series of nineteen mediastinal tumors which were operated upon, was able to determine the cellular nature of the pathology preoperatively in eleven cases. In seven cases the preoperative prediction of the cell type was erroneous, and an aneurysm was encountered in one patient. We are reporting a case of unusual interest in which a unique anomaly of the aorta was found which simulated a mediastinal tumor.

CASE REPORT

The patient, a 58-year-old male, was admitted to the Veterans' Administration Hospital, Richmond, Va., on April 1, 1946, with a history of frequent "colds," weakness, and occasional pains in the chest. There had been some slight weight loss. He had been told that he had heart disease and had been advised to limit his activities.

Physical examination revealed a man of small stature. There was generalized arteriosclerosis of the medium-sized vessels. No cardiac enlargement was present. The blood pressure was 172 mm. of mercury systolic and 110 mm. of mercury diastolic. Fundoscopy revealed some blurring of the disk margins and some accentuated vascular markings on the temporal side of the disk on the left. Right fundoscopy was normal. The electrocardiogram demonstrated left axis deviation. Routine laboratory work-up was negative.

Roentgenographic examination of the chest revealed normal lung fields, no enlargement of the heart, and a dense mass in the posterior mediastinum extending from the level of the seventh rib to the tenth rib. Its convex border bulged into the left hemithorax. An overexposed posterior-anterior view of the chest emphasized the density of the "tumor mass"

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(Fig. 1). A lateral view of the chest showed an ill-defined tumor with its convexity posterior (Fig. 2, *A*). Fluoroscopic examination and a roentgenogram of the barium-filled esophagus showed the distal one-third of this organ displaced anteriorly and to the left (Fig. 2, *B*). There was no erosion of the vertebral bodies. There was some question of pulsation of the mass during fluoroscopy, but kymographic studies were not available to confirm this observation. A study of the upper gastrointestinal tract with barium revealed a slight filling defect of the duodenal bulb. The cholecystogram was negative.

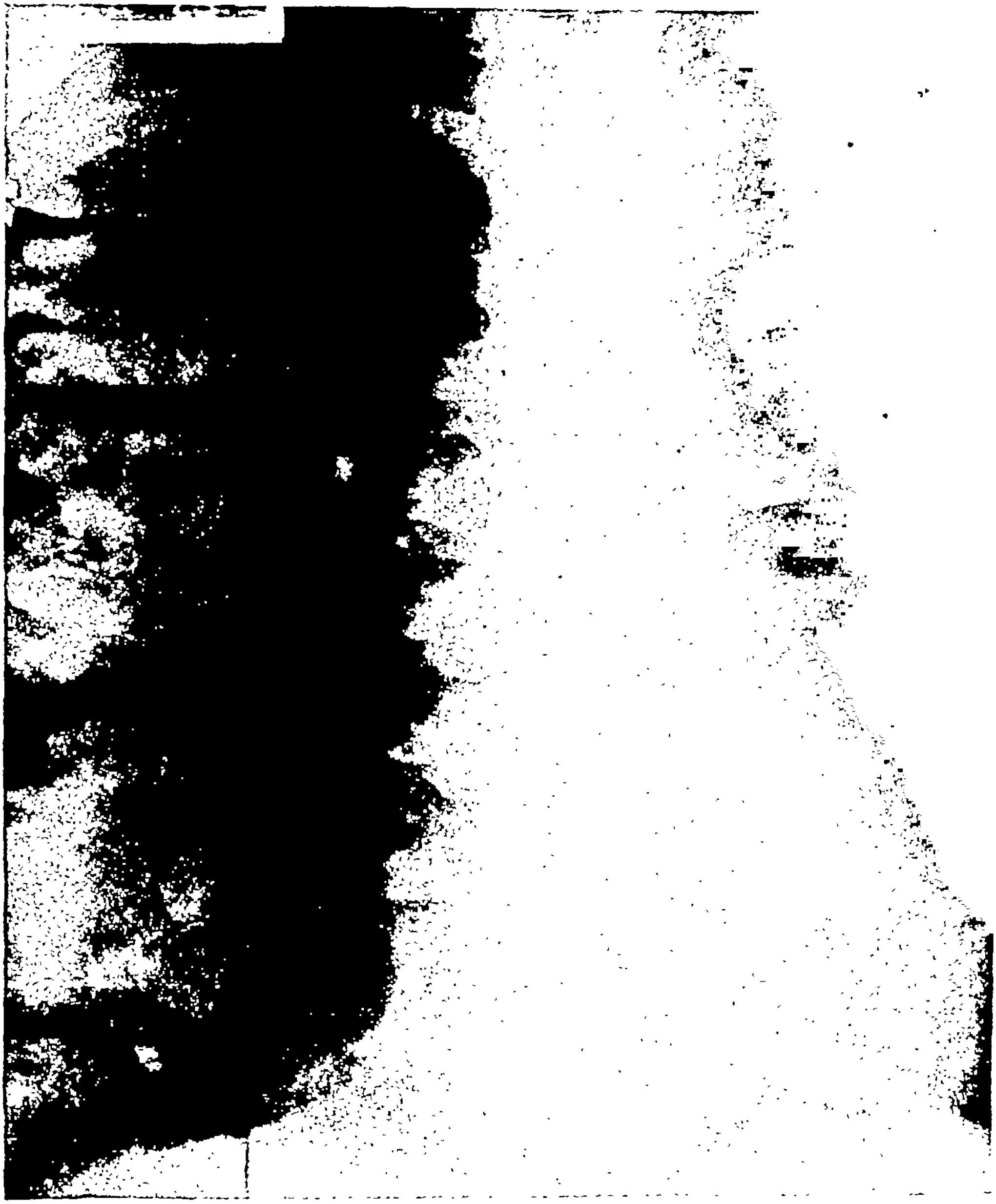

Fig. 1.—Overexposed posterior-anterior view of chest showing "tumor mass" extending from the level of the seventh rib to the tenth rib posteriorly.

The differential diagnosis was limited to a consideration of an aortic aneurysm and a soft-tissue mediastinal tumor. The anatomic location and the questionable pulsation favored the former, although saccular aneurysms of this portion of the aorta are seen infrequently. The absence of erosion of the vertebral bodies and the presence of an ill-defined mass with a posterior convexity extending into the costovertebral gutter (Fig. 2, *A*) made it necessary to consider mediastinal tumor.

On May 6, 1946, under endotracheal ether anesthesia, the left pleural cavity was opened through the periosteal bed of the resected eighth rib. The "tumor mass" was encountered adjacent to the angle of the resected rib. Further exploration revealed the mass to be an anomalous aorta. At the level of the seventh thoracic vertebra the arota deflected sharply to the left and took a horseshoe course before returning to the midline to pass through the aortic hiatus in the diaphragm (Fig. 3). The mediastinal pleura was opened in order to visualize the aorta to better advantage. There was marked arteriosclerosis of this vessel, but there was no increase in its circumference. The tortuosity which was most evident in the lateral plane was also present in the anterior-posterior diameter, thus accounting for the anterior displacement of the esophagus (Fig. 2, *B*). The left lung was normal.

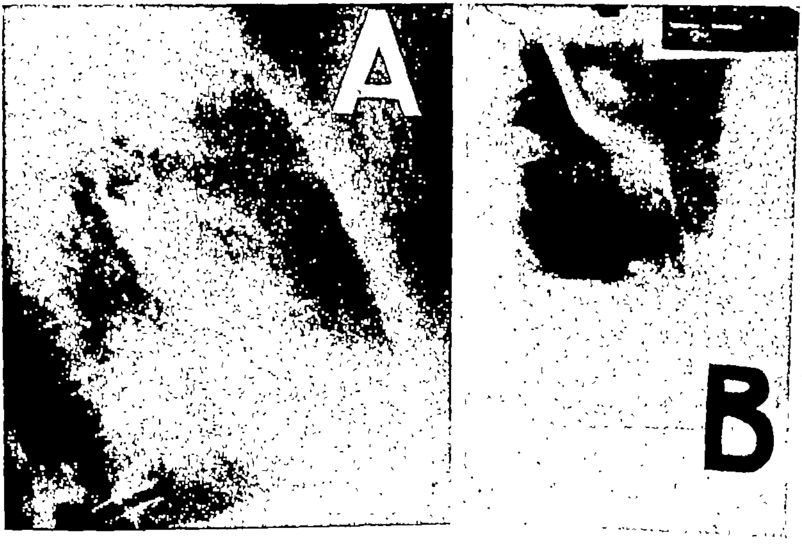

Fig. 2.—*A*, Left lateral view of chest "mass" ill defined. Note posterior convexity of tumor mass outlined on film. *B*, Spot film showing anterior displacement of barium-filled esophagus by tumor mass.

The patient's postoperative course was uneventful. On the twenty-third postoperative day he vomited approximately 300 c.c. of blood. This was attributed to the presence of a duodenal ulcer. At the time of his discharge from the hospital he was asymptomatic, although roentgenologic studies showed a persistent doudenal deformity. Three months following his discharge he had no complaints referable to either his gastrointestinal tract or thorax.

DISCUSSION

Tortuosity of the descending thoracic aorta producing deformity of the roentgen picture generally is believed to be produced by elongation of the adult aorta between its points of fixation at the heart and diaphragm. This picture is usually found in elderly individuals with hypertension and arteriosclerosis, although Khoo[2] has reported a case in a 12-year-old boy with a blood pressure of 110 mm. of mercury systolic and 60 mm. of mercury diastolic.

Rosler and White[3] have shown that tortuosity of the thoracic aorta produces deformity of the esophagus in the anterior-posterior plane, as did the case reported here. These authors give a particularly good description of the roentgenologic findings in cases with distorted thoracic aortae.

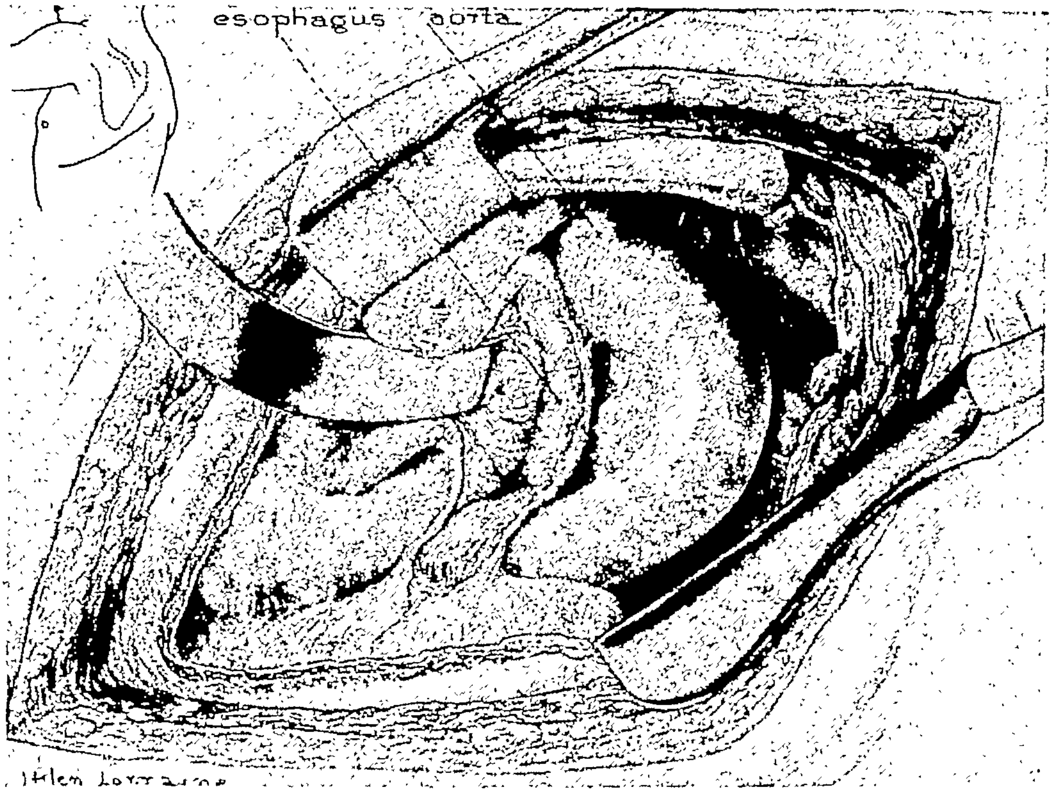

Fig. 3.—Reproduction of anomaly of aorta as seen at operation.

It seems unlikely that elongation in adult life sufficient to produce the extreme tortuosity found in this case could occur in the relatively short segment of aorta lying between the heart and the diaphragm. Assuming that this tortuosity was produced after the position of the heart and diaphragm had become fixed, it would necessitate almost a doubling of the length of the thoracic aorta. A congenital origin would seem more plausible. In early embryonic life the heart lies on the ventral aspect of the pharynx. During the later development of the neck, there is a gradual caudal migration of the heart into the thorax, which produces alterations in the ventral aorta and in the primitive aortic arches. On the right side, the fourth arch descends to the root of the neck, while on the left it is drawn within the thorax. It seems possible that alterations in this mechanism could produce tortuosity of any degree in the descending aorta. This would also explain the presence of tortuosity unaccompanied by either hypertension or arteriosclerosis in the case reported by Khoo.[2]

A second possible origin of the anomaly herein reported may be found in a study of the fusion of the paired dorsal aortae during early embryonic life. Congdon[4] has shown there is no duplication of the ventral aorta in man, but it is generally agreed that paired dorsal aortae exist. In embryos of about 3 mm. possessing twenty somites, there are two aortic arches and the dorsal aortae are beginning to fuse into a single descending aorta. This fusion takes place by the formation of medial communicating vessels between the two main trunks. These communications gradually coalesce, forming a single tube. If, during this process, a segment of the right or left aorta were obliterated between two communicating branches, the result would be a horseshoe-shaped curve similar to the one encountered in this case.

SUMMARY

A case of an anomaly of the thoracic aorta simulating mediastinal tumor has been reported. Two possible embryologic origins for this condition have been suggested.

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NERVE-BLOCKING THERAPY FOR FRACTURED RIBS

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THE application of regional nerve blocking to the therapy of disease or injury has most often been made with the primary purpose of controlling the associated pain. This was the object in utilizing paravertebral injections of analgesic solutions to the segmental nerves of fractured ribs as set forth in a preliminary report from this clinic.[1] A limited experience with thirteen patients at that time indicated that such a procedure was definitely useful in controlling pain. It was suggested also that the therapeutic value of such nerve blocking was considerable. Further experiences with 217 patients have served to substantiate the original contention that paravertebral injections of analgesic solutions are indicated to eliminate the pain of fractured ribs and are definitely useful to prevent more serious complications and reduce mortality.

Uncomplicated fractured ribs have little indication for nerve-block therapy other than to relieve pain. The patients are usually ambulatory and have simple therapeutic measures applied, such as adhesive strapping to maintain proper alignment of bone fragments and diminish movements of the involved chest wall. The associated pain is the most annoying complication during healing of the fracture. Such pain cannot be completely controlled by curtailing motion with strapping. Frequently the pain is not felt with respiratory movement only but is persistent or of the type often referred to as callus pain. Infrequently an intercostal neuralgia may accompany rib fracture. Regardless of the type of pain, its relief wins the patient's appreciation and makes it possible to apply desirable fixation of broken fragments without great annoyance. Complete pain relief can be obtained for the great majority of these patients by paravertebral nerve blocking, and the procedure can be depended upon to decrease pain regularly. This report does not substantiate this latter statement with results from extensive clinical experience. It has not been possible to follow accurately the outpatient group treated and the series is not large. However, the results with hospitalized patients with complicated rib fractures indicate that nerve blocking is desirable therapy for the pain of uncomplicated rib fractures.

The unfavorable sequence of chest pain, voluntary splinting of the thoracic wall, suppression of cough, and depression of pulmonary ventilation attendant upon the use of opiates in doses adequate for the relief of pain may predispose to the development of atelectasis, pneumonia, and even death, particularly in cases with extensive injury. According to Berry:[2] "It is generally recognized that contusions of the chest wall may cause a localized atelectasis of the underlying lung with bronchitis or pneumonia, or may activate a quiescent tuberculosis." He states further, "that the best prevention against atelectasis and pneumonia is sufficient sedation to allay pain on respiration and thus prevent too

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much splinting of the injured chest, but yet not enough to depress respiration." There can be little disagreement with the thesis proposed, but it is extremely difficult to maintain the precision balance between sedation which will just relieve pain and the depression of respiration and cough reflex which all too frequently ensue.

It cannot be denied that the effects of opiate administration are not associated with increased pulmonary ventilation but rather the reverse.[3] On the other hand, Harmon, Baker, and Kornegay[4] showed a marked increase in the vital capacity following procaine infiltration into the fracture sites, a phenomenon associated with the obtundation of pain.

In actual practice, the crux of the therapeutic problem involved in the pain of fractured ribs is analgesia which promotes tracheobronchial drainage and normal or increased pulmonary ventilation. Interruption of the painful stimuli by nerve block provides therapy for pain which, unlike the opiates or immobilization, assists in the restoration of normal function in the respiratory tract. Infiltration of the intercostal nerves with one of the local anesthetic agents enables the patient to cough freely without pain, permits the abandonment of voluntary splinting of respiratory movements and possibly releases the bronchial and bronchiolar spasm associated with the chest wall injury.[5] The need for rigid immobilization with plaster or adhesive strapping is eliminated. A fairly loose dressing which does not impede breathing may be useful for fixation in those cases where multiple anterior and posterior fractures have produced a "flail" chest with paradoxical motion.[6] However, it must be emphasized that regional analgesia for fractured ribs does not avoid the necessity of surgical intervention in those patients with severe intrapleural and thoracic visceral injury. It must be considered as a valuable adjunct in those instances. In patients upon whom operative interference is unnecessary, nerve block may be considered the primary therapeutic implement.

Intercostal nerve block has been recommended in the treatment of fractured ribs. Grieco[7] described a group of patients treated by alcohol injections of the intercostal nerves who had quick relief of pain and for whom the period of disability was greatly shortened. Physiologic section of an intercostal nerve with procaine solution is not a difficult procedure if comparatively large amounts of solutions are used, since the infiltration of tissues covers a wide area. It is considerably more difficult to inject alcohol in close contact with an intercostal nerve and, with the small amounts that should be used, denervation may be ineffective except at or very close to the point of injection. If large amounts of alcohol are employed, tissue destruction may be extensive and a complicating neuritis will be more frequent. Moreover, the injection of a single intercostal nerve does not always afford complete relief, since the innervation of the rib by accessory filaments from contiguous nerves is not interrupted. Paravertebral injections are to be preferred and it is recommended that in addition to the nerve corresponding to the fractured bone the next above and below be injected. The technique allows convenient treatment of all fractured ribs that are not complicated by injury to the vertebra or superficial injury at the site of skin puncture.

The technique for paravertebral injection of thoracic segmental nerves is essentially that of Labat[8] and has been described in detail in the original report by Rovenstine and Byrd.[1] The technique for injecting by the intercostal route is a simple one. It is performed by raising a skin wheal along the midaxillary line in the intercostal space below the rib involved. Through this wheal a 5 cm. needle unattached to the syringe is introduced and passed under the lower border of the rib in the direction of the intercostal nerve, and injection is completed with 3 to 5 c.c. of solution. Care must be taken to test for entrance into the pleural cavity by placing a drop of the solution over the opening in the needle. If the drop is sucked in, the pleural cavity has been entered and the needle must be withdrawn and re-inserted. Meticulous aspiration is mandatory to avoid intravascular injection. It is necessary in both methods to block the nerves immediately above and below the injured ribs to secure adequate anesthesia in the fracture site.

Infiltration directly at the site of fracture has been used to some extent but it has several disadvantages. The skin area immediately over the fracture may be contused and unfavorable for the introduction of a needle. Only small amounts of solution can be injected to bathe the involved nerve. The relief of pain with both local infiltration and nerve block by the intercostal route is less complete than following a paravertebral block.

The dangers consequent to the procedures discussed have been pointed out frequently and are attendant upon the possibility of entering the lung, the pleural cavity, a blood vessel, or the subarachnoid space. These accidents are uncommon, are easily recognized, and, therefore, should not produce serious complications. Careful aspiration will obviate injection into the lung tissue or a blood vessel because in both instances blood will be drawn back into the syringe. In addition the injection of procaine into the lung often causes coughing and regularly a metallic taste is noted by the patient. A further precautionary measure is slow injection at the start and, if coughing is induced, immediate cessation and reinsertion of the needle. In paravertebral blocks there is the possibility of injecting into the subarachnoid space either because of an abnormal prolongation of the dura along the nerve beyond the intervertebral foramen or by the introduction of the needle through the intervertebral foramen itself. In either case careful aspiration will demonstrate the presence of cerebrospinal fluid and reinsertion of the needle is indicated.

This report is based upon detailed examination of the results obtained in 217 patients with fractured ribs treated with nerve block at Bellevue Hospital over a period of seven years. This number does not represent the total number of fractured rib cases seen here during this period, but includes only those in whom pain was an important consideration. Some were recent fractures, others old with persistent pain. Many were complicated with other injuries and almost all had more than one rib involved. One hundred and ninety-nine patients were treated with paravertebral somatic blocks, ten with intercostal blocks and eight were given both (Table I).

Only one injection was required in more than 75 per cent of the cases to obtain relief of pain. This figure agrees well with the results of Harmon and associates[4] who report 25 per cent of their uncomplicated fractures required a second block. In the group receiving paravertebral blocks, 158 were blocked but once, thirty-six were blocked twice, four three times, and one required four injections. Of the patients receiving intercostal blocks, eight required but one block and two were repeated the second time. All the patients in whom both paravertebral and intercostal blocks were done required more than one injection. In all, a total of 280 blocks were completed (Table I).

TABLE I

TECHNIQUE	PARAVERTEBRAL SOMATIC	INTERCOSTAL	COMBINED	TOTAL
Case Incidence	199—91.7%	10—4.6%	8—3.7%	217
Frequency of Block				
One	158—79.4%	8—80%	0	
More than one	41—20.6%	2—20%	8—100%	
Number of Blocks	246—87.8%	12—4.3%	22—7.9%	280
Anesthetic Solutions				
Procaine	64—32.2%	4—40%	1—12.5%	69—31.8%
Procaine and alcohol	119—59.8%	4—40%	2—25%	125—57.6%
Others	16—8.0%	2—20%	5—62.5%	18—10.6%

A variety of solutions were used in the procedure. The most common was procaine (1 to 2 per cent), which in about two-thirds of the cases was followed by the injection of 1 to 2 c.c. of absolute alcohol (Table I). The other solutions used were procaine and ammonium chloride, intracaine, monocaine, and several experimental drugs. The results varied little. When procaine and alcohol was used, there was a slightly longer period of relief from pain than when procaine alone was injected. A single case of alcoholic neuropathy was noted, and there was one instance of painful regeneration of a nerve three months after alcohol block.

Fourteen of the patients had a single rib fractured. The preponderant majority (93.5 per cent) had from two to nine ribs involved and a third of the total number had other complications. Some of these were fractures of other bones, pneumonia, atelectasis, subcutaneous emphysema, hemothorax, pneumothorax, pleural effusion, and trauma of the abdominal viscera (Table II).

TABLE II

COMPLICATIONS PRESENT	NO. PATIENTS
Other Fractures	28
Pneumothorax	8
Pneumonia	16
Subcutaneous Emphysema	12
Atelectasis	2
Pleural Effusion	8
Lacerated Lung	3
Kidney Trauma	2
Chronic Bronchitis	2
Tuberculosis	3
Hemoptysis	3
Head Injuries	10
Laceration of Liver	1
Ruptured Spleen	1

The results in this series were graded as (A) good, providing marked pain relief and permitting the patient to cough freely; (B) moderate, pain relief incomplete but of appreciated value; and (C) poor, no relief obtained. A final classification of "questionable" embraces those cases in which there was a language barrier, an uncooperative patient, or insufficient data recorded. Eighty and six-tenths per cent were classed as good results, 11.9 per cent as moderate, 2.3 per cent as poor and 4.6 per cent as questionable (Table III).

TABLE III

		DEATHS		
		RELATED TO RIB FRACTURES	OTHER CAUSES	MORTALITY RATE
Uncomplicated Cases	142	—	—	—
Good	83.1%			
Moderate	11.3%			
Poor	0.7%			
Questionable	4.9%			
Complicated Cases	75	5	4	12%
Good	76.0%			
Moderate	13.3%			
Poor	6.7%			
Questionable	4.0%			
Total No. Cases	217		Gross Mortality	4.1%
Single Rib Fractured	14			
Multiple Ribs Fractured	203			

There was one death in the series which might be attributed to the procedure. This was a man who was admitted with fractured ribs (left, one to six), hemopneumothorax with paradoxical respirations, a head injury of undetermined extent, and shock. He had been treated successfully for shock for fourteen hours prior to the block. A paravertebral somatic block was completed on the left side (T1-7) with 1½ per cent procaine using a total of 30 c.c. Relief of pain was immediate and skin anesthesia on the left thoracic wall was limited to the area of the nerves blocked. He was sensitive to pain on the contralateral side and below the area blocked on the same side. Six or seven minutes after the block was completed, fibrillary twitchings occurred around his mouth and respirations suddenly ceased. His pulse was unobtainable and he became cyanotic. Endotracheal resuscitation was unsuccessful. Postmortem examination revealed multiple fractures of the ribs and a dilated right heart.

There were eight other deaths in the series, making the gross mortality rate 4.1 per cent (Table III). Three of the deaths were due primarily to pneumonia, one to pneumonia with laceration and contusion of the liver, one to pneumonia and subcutaneous emphysema of the entire body, and one to pneumonia and laceration of the brain with intracranial hemorrhage. One autopsy revealed an extensive pneumothorax with laceration of the lung. The eighth death in the series occurred in a woman who developed delirium tremens and died two days later. The mortality rate in this series is less than in the series of fractured ribs reported by Knoepp,[9] none of whose patients were treated by nerve block. In his series, all patients with uncomplicated cases survived but, when complications were present, mortality varied from 23 to 71 per cent depending upon the severity of the complications. In this series none of the uncomplicated cases ended in death but of the complicated ones only 12 per cent terminated in death.

In the majority of instances the relief of pain was *immediate* following the block and the patient was able to cough freely. Some of these had radiographic evidence of pneumonia processes with elevation of temperature, and within a day or two after a successful block remarkable clearing was demonstrated and the temperatures had returned to or near normal. The ability to cough and to ventilate more fully is responsible undoubtedly for such satisfactory recovery.

In addition to the patients with fractured ribs reported here, paravertebral and intercostal blocks have been used frequently to treat postoperative pain after a variety of thoracic and upper abdominal operations. Regularly, the relief of pain with its attendant freedom to cough and breathe without restraint has brought about considerable clinical improvement, and aided in securing a more normal convalescence. Gius[10] reports similar results in children who developed postoperative atelectasis following appendectomy.

SUMMARY

A survey of 217 patients with fractured ribs treated with nerve blocking, either paravertebral or intercostal, is presented. For more than 80 per cent of these, there was almost immediate relief of pain following the initial injection. Less than 25 per cent required more than one treatment. Among the patients presented, 93.5 per cent had multiple rib fractures and more than a third of the entire series had major complications associated with trauma. The mortality rate in the complicated cases was 12 per cent and the total mortality was 4.1 per cent. These results indicate that pain relief with nerve block is of significant value in preventing pulmonary infections and death of patients with fractured ribs.

This report was prepared under the supervision of E. A. Rovenstine, M.D.

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CONTINUOUS SPINAL ANESTHESIA FOR THORACOPLASTY

A REPORT OF 300 OPERATIONS

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THE introduction of continuous spinal anesthesia provided the surgical profession with new opportunities. Once this method was proved safe and free from complications, the possibilities were evident that procedures of greater magnitude could be carried out, since the duration of anesthesia could be maintained safely as long as necessary. In 1941, Lemmon[1] reported a series of 500 operations performed under continuous spinal anesthesia with no anesthetic deaths and no neurologic complications. Numerous reports in the last five years have confirmed the safety of the method.

This report, we believe, is the first to describe a series of major thoracic procedures performed under continuous spinal anesthesia. This series was begun in 1941, after a review of the pharmacology and the physiologic effects of procaine in the cerebrospinal fluid led to the conclusion that sufficiently high levels of anesthesia could be induced and maintained with continuous spinal technique to perform any stage of thoracoplasty, apicolysis, extrapleural pneumothorax, and similar procedures. This paper covers 300 thoracoplasties performed under continuous spinal anesthesia from February, 1941, to December, 1944.

PHARMACOLOGY AND PHYSIOLOGIC EFFECTS OF PROCAINE IN CEREBROSPINAL FLUID

Hatcher and Eggleston,[2] in 1916, demonstrated that the toxicity of Novocaine (procaine) given intravenously, depended largely upon the concentration of the drug and the rate of injection. Comparatively small doses given rapidly in high concentrations were fatal to animals, whereas large doses could be tolerated if given slowly and in low concentrations. They also showed that procaine was destroyed in the liver and not in the kidneys, as formerly supposed.

Bullock and MacDonald[3] showed in experiments on cats that the concentration of procaine in the cerebrospinal fluid falls rapidly from the point of injection where the concentration is highest, to the cisterna, where the concentration is negligible. Increasing the concentration, increased the duration of anesthesia. Only minimal amounts were found to be absorbed into the blood. The average amount found in the blood of human beings under spinal anesthesia was less than 0.002 mg., although as much as 300 mg. had been given intrathecally.

In 1942, Burford,[4] discussing the problem of absorption of procaine from the cerebrospinal fluid, concluded that the drug tended to become fixed to the immediately adjacent neural elements and that any absorption that took place was slow. The probable pathways of absorption are (1) through the spinal arachnoid villi and (2) through direct absorption

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from the capillary bed throughout the parenchyma after passage along the perivascular extensions of the subarachnoid space. He stated that "there is never a significant, still less a dangerous concentration of the drug in the blood."

After introduction into the subarachnoid space, the drug produces a progressive anesthesia from the sensory to the motor level. Experience has shown that small quantities of procaine suffice to maintain the desired depth of anesthesia once the initial block is established, since the greater part of the required dose is utilized in effecting the initial block.

Various explanations have been offered for the cause in the fall of blood pressure often seen during spinal anesthesia. This phenomenon has been attributed to decreased cardiac output, increase in the visceral capillary bed through trauma, handling of the intestinal tract, and to vasomotor paralysis.

Bradshaw,[5] in 1936, showed that the fall in pressure in experimental animals is due to vasomotor paralysis. He recorded the blood pressure of cats after the intrathecal injection of procaine and found the usual fall. After total sympathectomy there was no change in blood pressure after equal doses of spinal procaine. He concluded that the most important fibers concerned in the fall of blood pressure are those below the fifth thoracic segment. Bradshaw also stated that respiratory failure occurs only when high concentrations of procaine reach the region of the cervical cord and that this rarely happens in man except inadvertently. He suggested that the occasional collapse seen may be due to accidental injection of the drug into a dural vein. In this respect, the finding of Hatcher and Eggleston[2] should be remembered; namely, that procaine rapidly injected intravenously is extremely toxic. The problem in fall of blood pressure has been overcome in modern practice by the use of ephedrine or similar drugs prior to the induction of spinal anesthesia and the maintenance of circulating blood volume by the use of intravenous fluids during the course of operation.

A contribution to the problem of controlling spinal anesthesia was made by *Koster* and associates[6] when they showed that the duration of the paralysis of the lower extremities during spinal anesthesia could be shortened markedly by withdrawing cerebrospinal fluid. This observation, plus our own experience and that of others, shows that fixation of the drug in the neural elements can be but of short duration and that some other explanation must be given before we can understand anesthesia produced by procaine.

The fact that effective levels of anesthesia can be reached using much lower concentrations of procaine than originally recommended has definitely increased the safety of the anesthetic. We have progressively reduced the concentration employed to our present routine use of 2½ per cent solution.

In summary, the problems to be overcome in the use of (thoracic) spinal anesthesias were (1) fall in blood pressure, (2) toxicity of the agent employed, and (3) lack of control *over the injected drug. These have* been met by (1) the use of ephedrine and fluids to prevent and overcome the fall in blood pressure, (2) the use of procaine as a known safe agent, and (3) the use of continuous spinal as a method of controlling the level of anesthesia effectively for long periods of time.

CONTINUOUS SPINAL ANESTHESIA IN THORACIC SURGERY

It must be emphasized that spinal anesthesia has little, if any, place in intrapleural surgery. Here the anesthetic method of choice is inhalation anesthesia given through an endotracheal tube so that respirations can be controlled and bronchial secretions can be removed. However, in operations upon the chest wall, such as thoracoplasty, we believe continuous spinal anesthesia has advantages no other anesthetic offers.

The first patient in this series was operated on at Eagleville Sanatorium in February, 1941. The first patient at Jefferson Hospital was operated on by Dr. H. H. Bradshaw in March, 1941. From February, 1941, to December, 1944, 300 thoracoplasties were attempted under continuous spinal anesthesia. These operations were done on 114 patients, ages ranging from 19 to 59. For purpose of study, we have divided the cases into two groups (1) unilateral pulmonary tuberculosis and (2) bilateral pulmonary tuberculosis. A number of these patients have had pneumothorax maintained in the contralateral lung while undergoing staged thoracoplasty. Forty-nine patients had a Semb extrafascial apicolysis done in addition to the first stage thoracoplasty, an operation which adds substantially to the magnitude of the procedure.

DOSAGE

There is no arbitrary dose which can be advised. *Enough procaine is given to reach and maintain the desired level of anesthesia.* This amount varies greatly from stage to stage in the individual patient. The average initial dose has been 100 mg. of procaine and the total dose has varied from as little as 150 mg. to as much as 1,000 mg. It must be emphasized that the amount required is unpredictable. However, the majority, or 65 per cent of the stages, have been done with a total dosage of procaine ranging from 200 mg. to 400 mg. It should be observed at this point that with the solution now used, 2½ per cent procaine in spinal fluid, there is apparently very little danger of untoward effects on respiration, even when the level reaches higher than is desired. It is known that higher concentrations of procaine are needed to cause motor paralysis than sensory anesthesia. Apparently the dilution of procaine in the spinal fluid is such that it reaches the cervical region in amounts insufficient to cause paralysis of the diaphragm while concentrated enough to cause sensory loss and provide muscular relaxation. We have seen many patients with complete anesthesia of the scalp and tongue with no sign of respiratory difficulty.

EFFECT ON PULSE RATE, BLOOD PRESSURE, AND RESPIRATION

There was no appreciable difference in the effect of spinal procaine on the pulse rate in either the unilateral or bilateral group. Of the unilateral patients, 11⅓ per cent and of the bilateral patients, 15 per cent showed a fall in pulse rate at the conclusion of the operation. A rise in pulse rate averaging twenty-five per minute was shown in 28⅔ per cent of the unilateral and 27⅔ per cent of the bilateral cases. There was no change in pulse rate in 5⅔ per cent of the unilateral and 5⅓ per cent of the bilateral cases. In the remaining 1⅓ per cent of the unilateral and 1⅔ per cent of the bilateral cases, complete records were not kept.

As in pulse rate, there was no significant difference in the change in blood pressure in each group. A fall in blood pressure was shown at the conclusion of the operation in 30⅓ per cent of the unilateral and 35⅓ per cent of the bilateral cases. A rise in blood pressure was noted at the termination of the surgical procedure in 6⅔ per cent of the unilateral and 5⅔ per cent of the bilateral cases. In 2 per cent of the unilateral and 1⅔ per cent of the bilateral cases there was no change in blood pressure. In the remaining 8 per cent of the unilateral and 7 per cent of the bilateral cases the desired information was not recorded.

In 29⅓ per cent of the unilateral and 33⅓ per cent of the bilateral cases there was a slight rise in respiratory rate, averaging four per minute. In ⅔ per cent of each group there was a fall in respiratory rate and in 12⅓ per cent of each group no change in respiratory rate was noted. In the remaining 4⅔ per cent of the unilateral and 3⅓ per cent of the bilateral cases, the necessary data was not recorded.

In ⅓ per cent of the *entire* group of unilateral cases and 3 per cent of the *entire* number of bilateral cases, operation was discontinued, for reasons stated later, as tabulated below. There was one patient with temporary arrest which responded promptly to oxygen, withdrawal of spinal fluid, and artificial respiration. There was one anesthetic death which will be discussed shortly.

TABLE I

	PULSE				BLOOD PRESSURE				RESPIRATIONS				DISCONTINUED OPERATIONS
	FALL	RISE	NO CHANGE	?	FALL	RISE	NO CHANGE	?	FALL	RISE	NO CHANGE	?	
Unilateral													
141 cases (47%)	34	86	17	4	91	20	6	24	2	88	37	14	1
Percentage	11⅓	28⅔	5⅔	1⅓	30⅓	6⅔	2	8	⅔	29⅓	12⅓	4⅔	⅓
Bilateral													
149 cases (49⅔%)	45	83	16	5	106	17	5	21	2	100	37	10	9
Percentage	15	27⅔	5⅓	1⅔	35⅓	5⅔	1⅔	7	⅔	33⅓	12⅓	3⅓	3

SUPPLEMENTARY ANESTHESIA

In 35 patients it was necessary to use one or more types of supplemental anesthesia. The percentage of operations thus requiring supplemental anesthesia was 11.7, the effectiveness of continuous spinal anesthesia in this series being 88.3 per cent. When the 10 cases which were discontinued are added to this group, the total effectiveness of the continuous spinal anesthetic method was 85 per cent.

COMPLICATIONS

Aside from the ten patients on whom operations had to be discontinued before completion, nine having bilateral lesions, there was no appreciable difference in the incidence of complications in the two groups. This is a very difficult subject to discuss and evaluate since we are attempting to separate the complications of the anesthetic from those inherent in the operative procedure itself. In many cases the differentiation cannot be made.

Of the operations, 4⅓ per cent were followed by nausea and 13 per cent by vomiting. This compares very favorably with the incidence of these phenomena following general anesthesia. While we are not able to furnish the figures as to the incidence in the cases done under cyclopropane and ether, it is our impression that nausea and vomiting to some degree followed the great majority of cases.

Postoperative headaches occurred in 9.3 per cent of cases. In none of these was the headache alarming or persistent. The percentage of headaches found in our cases was larger than in most reported series of infradiaphragmatic procedures done under continuous spinal anesthesia. However, we feel that this minor complication is a small price to pay for the many compensating advantages of the method.

Two patients, or 0.66 per cent went into a state of shock or collapse following *continuous spinal*, but responded to the usual methods of resuscitation. Whether this can be attributed to the anesthetic method is very questionable, since a far greater number was seen when inhalation anesthetics were being used.

In ten cases, it was necessary to discontinue the operation because of difficulties with the anesthetic or unsatisfactory condition of the patient. Although it is quite likely that some of these patients would have presented the same difficulties under inhalation anesthesia we are listing them as failures of the method. The following are brief summaries of the ten discontinued cases:

Case 1. Unilateral pulmonary tuberculosis. Anesthetic unsatisfactory since free flow of spinal fluid could not be obtained.

Case 2. Bilateral pulmonary tuberculosis. Free flow of spinal fluid obtained only after third puncture. Fluid was bloody. Satisfactory level was not obtained after administration of 500 mg. of procaine.

Case 3. Bilateral pulmonary tuberculosis. Satisfactory level was not obtained after 500 mg. of procaine had been given.

Case 4. Bilateral pulmonary tuberculosis. Patient had respiratory arrest during course of operation. Prompt recovery with oxygen, artificial respiration, and withdrawal of spinal fluid.

Case 5. Bilateral pulmonary tuberculosis. Marked cyanosis developed and operation was discontinued.

Case 6. Bilateral pulmonary tuberculosis. Patient had alarming rise in pulse rate and marked venous oozing. Extreme indrawing of intercostal spaces present after exposure of costal cage. Similar experience with this patient later, using regional block anesthesia with 1 per cent procaine solution.

Case 7. Bilateral pulmonary tuberculosis. Anesthesia could not be obtained above level of third thoracic vertebra.

Case 8. Bilateral pulmonary tuberculosis. Patient's blood pressure fell to 60/40 following skin incision and mobilization of scapula.

Case 9. Bilateral pulmonary tuberculosis. Anesthetic level satisfactory but patient was extremely restless and uncooperative. This case may represent one of untoward effects of premedication with barbiturates.

Case 10. Bilateral pulmonary tuberculosis. Anesthetic level satisfactory but patient restless and apprehensive. Very similar to Case 9.

MORTALITY

There was one anesthetic death in this series of 300 operations. This occurred early in our experience, in 1941. This patient was a white male, aged 37. Examination revealed bilateral pulmonary tuberculosis with extensive disease in the left lung and old fibrotic lesions in the right upper lobe. Sputum averaged 35 c.c. daily and was highly positive for tubercle bacilli. First stage thoracoplasty was carried out uneventfully on Nov. 10, 1941. On December 19, patient was scheduled for a second stage thoracoplasty. The spinal tap was bloody and a free flow was not obtained. These two contraindications to spinal anesthesia were ignored by the surgical team of the day and the patient was given 150 mg.

of procaine in a 5 per cent solution. Barbotage was used to extend the anesthetic solution upward. Two subsequent doses of procaine were given in 150 mg. and 100 mg. doses, disregarding the usual dosage of 40 to 60 mg. at two-minute intervals.

Oxygen was not given before cyanosis and restlessness were observed.

One hour after induction of the anesthesia and almost immediately after the last dose of 100 mg. of procaine was given, respiratory arrest occurred, followed momentarily by cardiac arrest.

Spinal fluid could not be withdrawn until a puncture at another level was made.

Artificial respiration with 100 per cent oxygen administration brought no response.

We believe that had the rules for the anesthetic been adhered to this fatality would not have occurred.

TECHNIQUE

Enough barbiturate is given the evening before operation to ensure a restful night. Two hours prior to operation 1½ grains of Nembutal or Seconal is given, and one hour prior to operation the patient is given ¼ grain of morphine sulfate and 1/150 grain of scopolamine, hypodermically. The patient is brought to the operating room with as little disturbance as possible. Many patients are drowsy but can be aroused easily. The patient is placed on the table in the position to be maintained during operation, the lateral decubitus. Ephedrine sulfate, ⅜ grain, is given hypodermically and a skin wheal is raised in the lumbar region over the widest interspace. Choice of interspace is unimportant. A small skin incision is made and the introducer is inserted through the interspinous ligament. A No. 19 gauge malleable needle is used. If bloody fluid is obtained, the operation is postponed. Upon entering the subarachnoid space the spinal fluid pressure is taken, after which a syringe is connected to the needle and it is determined whether or not a free flow of spinal fluid is present. This is highly important, since safe continuous spinal anesthesia for thoracic procedures cannot be carried out if a free flow of fluid is not present. Should the flow be unsatisfactory, a second puncture is made at another interspace.

When a free flow of fluid has been obtained, the malleable needle is bent so that it is parallel to the skin and then fixed with adhesive tape. A 30-inch rubber tube, with a volume capacity of 2 c.c. is then attached to the needle. Spinal fluid, 20 c.c., is then drawn into a syringe containing 500 mg. of procaine.

The initial dose is usually 100 mg. The table is then placed in a 15° Trendelenburg position. Subsequent 50 mg. doses of procaine are given at two-minute intervals until the desired level of anesthesia is obtained. The rate of ascent is usually two or three interspaces with each 50 mg. dose. It is rarely necessary in a first stage thoracoplasty to give more procaine after the level has reached the upper border of the scapula. While the patient is prepared and draped, the anesthesia level will rise to the angle of the jaw, the desired point to which anesthesia must rise for a first stage procedure and must be maintained at this level until closure of the upper angle of the incision has progressed to the midscapular area. At this stage of the operation the stopcock on the tubing is opened and spinal fluid is allowed to flow into the syringe. The amount of fluid removed varies with each patient and is dependent on the degree of sensation and motor function regained.

Removal of spinal fluid is discontinued if abdominal respiratory movements are present, if the patient can move the toes, or if the sensory level is in the midscapular area, at the conclusion of the operation.

The needle is not removed from the subarachnoid space until this degree of motor function or sensation has been restored.

Occurrence of nausea and retching is very uncommon since we discontinued barbotage and a 5 per cent solution, in 1942. If these symptoms do occur it is during induction or early in the course of the operation and are promptly relieved by withdrawal of spinal fluid; usually 2 to 4 c.c. will suffice. After a two- or three-minute interval, small doses of the drug are injected as needed.

Oxygen by mask is given to all patients as soon as the operation is begun.

ADVANTAGES

During an operation under continuous spinal anesthesia, there is excellent muscular relaxation. Mobilization and retraction of the scapula is facilitated. Respirations are quiet and not labored and paradoxical motion of the lung is minimized. Cyanosis is unusual and the shock so often seen when the patient is moved from the operating table after inhalation anesthesia is almost nonexistent.

These patients require much less nursing care than those operated upon under inhalation anesthesia. Recovery is prompt and uneventful. Secretions in the bronchial tree are much less likely to be aspirated into the contralateral lung since at no time after operation is there loss of the cough reflex.

Patients in both hospitals requiring thoracoplasty prefer the continuous spinal method of anesthesia. The patients who have had previous stages done under inhalation anesthesia and subsequent ones under spinal anesthesia are unanimous in their preference for spinal anesthesia because of the comfort during operation and the prompt, uneventful recovery.

CONCLUSIONS

1. Continuous spinal is a safe anesthetic for extrapleural thoracic procedures.
2. The operative procedure is made easier because of quieter breathing, better muscular relaxation, and lessened paradoxical motion of the lung.
3. Recovery is prompt, shock is rare, and postoperative nursing requirements are reduced to a minimum.
4. Postoperative complications are fewer than those occurring after inhalation anesthesia for similar operative procedures.

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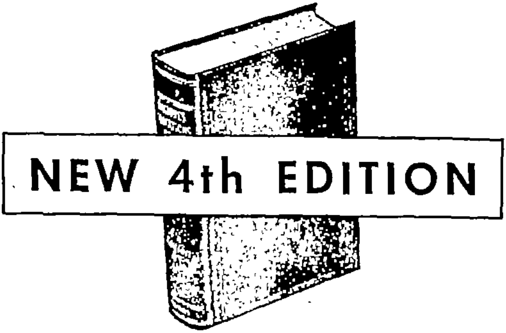

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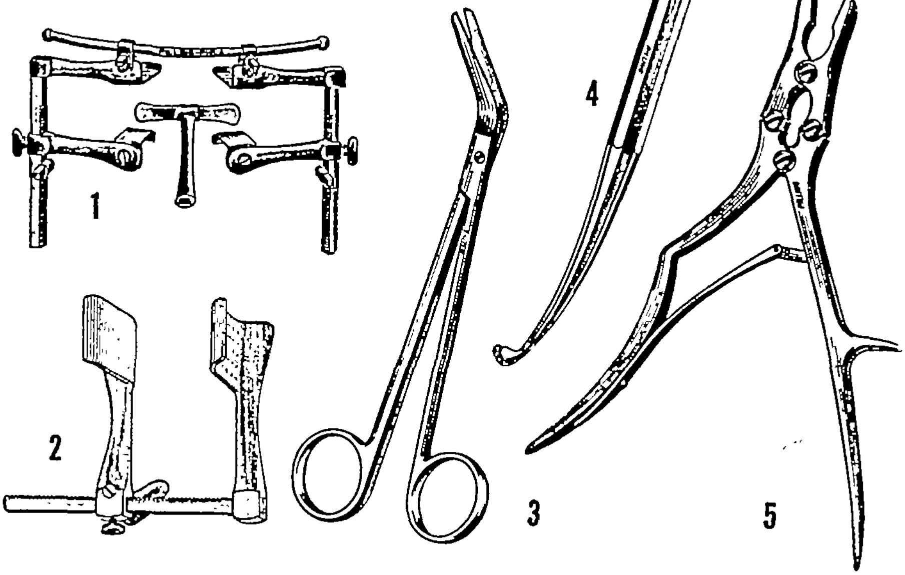

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Original Communications

SURGICAL LESIONS OF THE ESOPHAGUS SEEN IN AN ARMY THORACIC SURGERY CENTER

EARLE B. KAY, M.D.
CLEVELAND, OHIO

DURING the past three years, 133 patients with diseases and injuries of the esophagus have been treated at this army thoracic surgery center. This series represents mainly the nonmalignant conditions, for very few patients in the younger age group have carcinomas. The purpose of this paper is the presentation of the treatment employed. Forty-two esophageal operations were performed by the staff with only one operative mortality, an incidence of 2.4 per cent. This mortality occurred following the local excision of a benign tumor. The other patients were treated by instrumental dilatation, conservative measures, or did not require treatment. The esophageal conditions treated and the operations performed are tabulated in Tables I and II. No attempt will be made to discuss the preoperative and postoperative management. The potential dangers of esophageal surgery associated with the inherent anatomic characteristics of the esophagus, the virulence of the bacterial flora resulting from stasis, and the poor general condition of the patient caused by his inability to eat were well appreciated and adequate preoperative measures were employed.

STRICTURE

Twenty-seven patients had cicatricial stenoses, fourteen of which were due to consumption of a caustic fluid. In eleven of these the caustic was contained in sabotaged alcoholic beverages and consumed accidentally overseas. The other three resulted from suicide attempts. Six patients had stenoses secondary to war wounds of the esophagus. A congenital short esophagus was complicated by ulceration, scarring, and stenosis at the esophagogastric junction in three patients. Three were secondary to an esophagitis. One resulted from local ulceration and constriction due to a foreign body.

From the Section of Thoracic Surgery, Kennedy General Hospital, Memphis, Tenn.

Read at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

TABLE I. CLASSIFICATION OF 133 ESOPHAGEAL CONDITIONS SEEN IN A THORACIC SURGERY CENTER

TYPES	NO.		TREATMENT	NO.	
Strictures:					
Caustic	14		Intrathoracic esophagoplasties	4	
Congenital short esophagus	3		Antethoracic esophagoplasty	2	
Traumatic	6		Repeated esophageal dilatations	21	
Esophagitis with stenosis	4				27
		27			
Tumors:					
Benign: neurofibroma	1		Local excision	3	
leiomyoma	2		Resection with anastomosis	2	
Malignant: adenocarcinoma	4		Inoperable (exploratory)	3	
squamous cell carcinoma	1		Total gastrectomy, transverse colectomy and esophagojejunostomy	1	
adenocarcinoma (stomach)	1				9
		9			
Cysts:					
Dermoid	2		Transthoracic esophageal resection with esophagogastric anastomosis	1	
Simple	2				
Bronchogenic	1		Local excision	4	
		5			5
Diverticulum:					
Traction	4		No treatment indicated	8	
Pulsion	3		Transpleural diverticulectomy	1	
Epiphrenic	2				9
		9			
Traumatic injuries, war wounds:					
Tracheoesophageal fistula	1				
Esophagocutaneous fistula	3				
Mediastinal and paraesophageal abscesses	6		Drainage of paraesophageal abscesses	5	
Paraesophageal shell fragments	8		Removal of shell fragments	6	
		18			11
Varices:		1	Too extensive for treatment		
Cardiospasm:		20	Cardioplasties	11	
			Dilatations	9	
					20
Congenital short esophagus:					
Carcinoma	1		Resection with anastomosis	1	
Stricture	3		Required dilatations	3	
Ulcer	4		Temporary neuremphraxis	3	
Essentially asymptomatic	6		Fixation of diaphragm above esophagogastric junction	1	
Symptoms improved by modified ulcer regimen	6		No surgical treatment	12	
		20			20
Hiatal hernia:		9	Required repair of hernias	5	
Paraesophageal hernia:		4	Required repair of hernias	1	
Extrinsic esophageal obstruction: (tumors, cysts, aneurysms, tuberculous nodes)		11	No treatment directed to the esophagus		
Total		133			

Instrumental dilatation over a previously swallowed thread as a guide was effective in twenty-one patients. This was impossible or ineffectual in six patients and surgical therapy was employed. The degree of stricture in three patients was too great to allow instrumentation. Two others had upon admission strictures associated with mediastinitis and multiple fistulas incurred accidentally during previous attempts at instrumental dilatation. Instrumentation

TABLE II. ESOPHAGEAL OPERATIVE PROCEDURES

TYPE	NO.	MORTALITY
Cardioplasty	11	0
Reconstructive transthoracic resection and esophagogastric anastomosis	7	0
Reconstructive antethoracic esophagodermatojejunal anastomosis	2	0
Transthoracic total gastrectomy, transverse colectomy and esophagojejunostomy	1	0
Esophagostomy	2	0
Transthoracic diverticulectomy and fixation of diaphragmatic hiatus above esophagogastric junction (congenital short esophagus)	1	0
Excision of intramural tumors and cysts	7	1
Drainage of paraesophageal abscesses	5	0
Removal of paraesophageal foreign bodies (shell fragments)	6	0
Total	42	1

in another patient was possible but not effective. Reconstructive operations were necessary in these six patients. Four intrathoracic esophagoplasties and two antethoracic jejunodermatoesophagoplasties were performed. The third stage in one of the latter patients has not been performed as yet. For the intrathoracic esophagoplasties, a tube was fashioned from the greater curvature of the stomach. The blood supply for this tube was furnished primarily by the right gastroepiploic artery. One of the intrathoracic esophagoplasties was total, the anastomosis being just within the thoracic inlet; another was approximately one inch below the thoracic inlet, and the other two were at the level of the aortic arch.

For contemplated intrathoracic esophagogastric anastomosis near the thoracic inlet, adequate exposure was felt to be best obtained by using first an abdominal incision for the mobilization of the stomach and construction of the gastric tube and then a high thoracic incision for formation of the anastomosis. When the contemplated anastomosis is at the level of the aortic arch or below, adequate exposure can be obtained by a single transthoracic approach.

The antethoracic esophagoplasties were performed in three successive stages at three-week intervals. In the first patient the esophagus was transected transthoracically just proximal to the stricture and brought out through a cervical incision as an esophagostomy. The second stage consisted of formation of a jejunal tube. Sufficient jejunum was mobilized to reach easily to the neck to allow esophagojejunal anastomosis, but during the preparation of inserting the jejunal tube beneath the skin of the chest wall one of the primary arcuate arteries was torn and eight inches of the jejunum had to be sacrificed. The esophagus and the jejunum were then joined by a skin tube at the third stage.

The functional results following both the intrathoracic and antethoracic esophagoplasties have been entirely satisfactory. The patients are able to eat regular diets with no difficulty, although the patients with the antethoracic esophagoplasty must chew their food more thoroughly and more time is required for the passage of food through the tube.

If a reconstructive operation is contemplated, and the condition of the patient is such that supplementary feedings are necessary, the decision as to a gastrostomy or jejunostomy should rest with the surgeon performing the latter operation. An inconveniently located gastrostomy may prevent formation of a tube from the stomach. A jejunostomy is preferred if an intrathoracic anastomosis is contemplated.

TUMORS

There were three benign, intramural, esophageal tumors, two leiomyomas and one neurofibroma. All three arose between the submucosa and muscularis and all were removed by local excision. The mural attachment of one large leiomyoma of the upper third of the esophagus was so extensive that considerable dissection of the esophageal wall was necessary. Though this appeared viable at the termination of the operation, necrosis occurred which resulted in mediastinitis and empyema. This patient died on the fifth postoperative day.

There were five esophageal carcinomas, of which only two were operable. These patients were treated by esophageal resection and esophagogastric anastomosis. One of these patients is of interest as the lesion was an adenocarcinoma arising at the esophagogastric junction in a patient having a congenital short esophagus. A sixth patient had a large fungating carcinoma of the stomach that had infiltrated the lower esophagus, the spleen, and the transverse mesocolon. A total gastrectomy, splenectomy, and transverse colectomy were performed, followed by an esophagojejunostomy and double-barrel colostomy.

CYSTS

There were five unusual and interesting esophageal cysts. All of these were intramural and arose between the submucosa and muscularis. There were two dermoid cysts, two simple cysts, and one bronchogenic cyst. Local excision was possible in four of the five cysts, but resection of the lower esophagus including the cyst followed by an esophagogastric anastomosis was necessary in the fifth. Whenever possible, cysts and benign tumors are explored through the left pleural cavity, for if esophageal resection is found advisable, the stomach is readily available for anastomosis.

DIVERTICULA

There were four traction, three pulsion, and two epiphrenic diverticula. The traction diverticula were thought to be secondary to tuberculous mediastinitis and mediastinal lymphadenopathy in that large calcified mediastinal nodes were present in all cases. The orifices of the diverticula could be seen during esophagoscopic examinations. The mucous membrane about the orifices appeared taut but not inflamed or ulcerated. In two cases, retained barium could be demonstrated within the diverticula for several days following radiographic examinations. Faulty emptying was probably responsible for the symptoms present. In view of the fibrous mediastinitis and calcified mediastinal lymphadenopathy, no treatment directed to the esophagus was indicated.

The three pulsion diverticula of the thoracic esophagus were associated with vague complaints consisting mainly of dysphagia and night pain. No esophageal obstruction was noted roentgenographically. Night pain may have resulted from accumulation of gastric secretion in this pouch when the patient was in the prone position. The symptoms in this group were not severe nor consistent and were improved by conservative measures including feedings or amphojel prior to retiring.

The two patients with epiphrenic diverticula had congenital short esophaguses. Both of these patients complained of epigastric and substernal pain occurring several hours after meals and especially at night. One patient also had considerable pain upon swallowing. Esophagoscopic examinations demonstrated one of the diverticula to be just below the esophagogastric junction and the other just above it. There was considerable inflammation and erosion of the mucous membrane immediately around the orifices of the diverticula. One patient was completely relieved of symptoms by a Sippy regimen and the other required excision of the diverticulum and suture of the diaphragm at a higher level about the esophagus before symptomatic relief was obtained.

TRAUMATIC INJURIES OF THE ESOPHAGUS

Traumatic injuries of the esophagus were seen in ten patients. Seven were caused by shell fragments or bullets and one by a bayonet. Two resulted from instrumental perforation during esophagoscopy elsewhere. Five of these esophageal injuries were associated with injuries of the trachea necessitating emergency tracheotomies and gastrostomies. Six patients had developed paraesophageal and mediastinal abscesses which required drainage. Residual abscesses and esophagopleural and esophagocutaneous fistulas were present in these patients upon admission. Most of these latter patients required secondary drainages and the fistulas persisted for a number of months. Another patient had an esophagotracheal fistula that healed on conservative management employing chemotherapy.

It was possible to remove the tracheotomy and gastrostomy tubes soon after the patients returned to this country. Six patients developed localized esophageal strictures that responded readily to instrumental dilatation. Two additional patients, as previously described, had upon admission esophageal strictures associated with fistulas resulting from instrumental dilatation.

Large shell fragments or bullets were in contact with the esophagus in eight patients. Two of these had caused localized fistulas or diverticula. It was felt advisable to remove the metallic foreign body in six of these patients.

ESOPHAGEAL VARICES

Only one patient with esophageal varices was seen. He had had a splenectomy five years previously with apparent symptomatic relief from bleeding. During the month prior to admission three massive hemorrhages occurred. Radiographic studies revealed extensive varices the entire length of the esophagus. The extensiveness of the varices precluded treatment.

CARDIOSPASM

Twenty patients with cardiospasm were treated. In eight patients with moderate degrees of cardiospasm satisfactory symptomatic improvement resulted from instrumental dilatation though there was no evidence of reduction in the size of the esophagus afterward.

Transpleural cardioplasties were performed in eleven patients not significantly improved by instrumental dilatation. In contrast, the operative group were given more permanent and complete relief of symptoms and showed striking reductions in the size of the esophagus postoperatively. One patient is now awaiting operation.

All of the cardioplasties were performed through a transpleural approach, for it was felt that this allowed better exposure of the esophagogastric junction, better mobilization of the lower esophagus and cardia of the stomach and, in turn, the construction of a larger aperture than would be possible through an abdominal approach. The new stoma was made large enough to allow for reduction in size as the caliber of the esophagus decreased. Its size determines the ease with which the esophagus empties itself and the likelihood of its reduction in caliber. The first three cardioplastics were performed in a manner similar to a Finney type of pyloroplasty technique, and the others in a manner similar to a Heineke-Mikulicz type. The latter was found to be the easier and more satisfactory procedure.

CONGENITAL SHORT ESOPHAGUS

Twenty-three patients having a congenital short esophagus were observed. The presenting symptoms in most instances were dysphagia, painful swallowing, substernal pain, vague epigastric pain and discomfort, and night pain. One patient had a carcinoma at the esophagogastric junction, three a high-grade stricture, six an ulcer, and two were associated with epiphrenic diverticula. The others were uncomplicated. The patient with the carcinoma had an esophageal resection and esophagogastric anastomosis. The strictures responded to instrumental dilatation and a modified Sippy regimen. Three patients had temporary phrenic neuremphraxis with only partial or temporary improvement. One of the patients with an epiphrenic diverticulum was completely relieved by means of a Sippy diet. The other one was not significantly improved by this measure. The dysphagia, painful swallowing, and night pain persisted. These symptoms disappeared following a transpleural diverticulectomy and resuturing of the diaphragm to the esophagus at a higher level. The others were relieved of symptoms by carefully following a modified Sippy regimen.

Patients with a congenital short esophagus may have retention of gastric juice in the gastric pouch between the diaphragm and esophagogastric junction. This is more likely to occur when the patient is in the prone position while sleeping. The presence of the retained gastric juice may allow peptic digestion of the lower esophageal mucous membrane and be responsible for the high incidence of ulcers. Such ulcers can frequently be detected by esophagoscopic examinations when not apparent on esophagrams. Sleeping in the semiprone position may aid in emptying of the gastric juice and allay symptoms. Feedings, amphojel, or some buffer substance should be taken prior to retiring to buffer the gastric acidity.

A congenital short esophagus is occasionally misinterpreted as being a hiatal hernia, and in some patients both factors may be present. In the former condition the esophagus is taut as well as that portion of the stomach above the diaphragm, whereas in the latter condition the esophagus and stomach appear redundant.

SUMMARY

The conditions found and the treatment employed at this Army Thoracic Surgery Center in 133 patients with various lesions of the esophagus are presented. There were forty-two major esophageal operations performed by the Staff with one mortality, an incidence of 2.4 per cent. The other patients were treated either by instrumental dilatation or by conservative measures.

DISCUSSION

DR. R. H. SWEET, Boston, Mass.—This is a perfectly marvelous series of cases, and the results are certainly excellent.

This is a field in which I am particularly interested, and the paper brought to mind questions about various matters which have to do with these problems, many of the points, of course, being still controversial.

For instance, there is the matter of the incision. I have performed five esophagectomies for these stricture cases, pulling the stomach all the way up to the apex of the chest for an anastomosis. In all of these, a single thoracic incision was used. Dr. Kay does not mention whether he removed the esophagus or not. He merely speaks of it as ''esophagoplasty.'' I would like to have him clarify that point, having felt that one should take the esophagus out below the point of anastomosis. I have always done it through a single thoracic incision.

Even though we have all had successes, such as the young man whom he showed in the pictures, external esophagoplasty is never a thoroughly satisfactory procedure, and I feel that something else better than that should be substituted, if possible.

One question, when it comes to that: Is there any evidence in Dr. Kay's experience that anastomosis or at least sutures between skin tubes and the jejunum or the stomach tend to develop strictures? They seem to do so, whereas, on the other hand, if we suture the esophagus to the skin, they do not occur. The reason for that may be that the esophageal mucosa is so much more like the skin than is the jejunal or gastric mucosa.

Then the enormous subject of cardiospasm comes to the fore. I would like to say that I have tried about every possible surgical maneuver, including the one which he describes, with excision and various cardioplastics of one sort or another. Excluding, of course, the fact that many cases of cardiospasm do not need surgery, the important point is: How long have these cases been followed? After a good deal of early encouragement, we are beginning to see some difficulties arising during the late convalescence, months and years following the procedure. In that group of cases, experience seems to favor the Heineke-Mikulicz type of procedure, of longitudinal incision and transverse suture. Results in the direct surgical approach to cardiospasm have been best when that method was used.

DR. FREDERICK R. HARPER, Denver, Colo.—In 1942, Dr. Lewis of Philadelphia and I worked on some cadavers in order to discover a suitable approach to the upper abdominal organs through the chest. We decided that an approach which involved removal of the ninth rib with extension of the incision into the abdomen and splitting the diaphragm would give the least exposure. Since I returned from overseas, I have had an opportunity to use the incision for removal of carcinomas of the cardiac end of the stomach and esophagus and also for the repair of diaphragmatic hernias. The incision has proved to be entirely satisfactory.

(Slide.) The incision is made over the ninth rib and extended for about two inches into the abdomen. The ninth rib is removed and the chest entered through its bed. The costal arch is then divided and the incision is extended for about two inches into the abdomen. The pulmonary ligament is then divided and after that the diaphragm is split, starting at the divided costal arch and extending to the esophageal hiatus. This gives exposure of the entire esophagus below the aortic arch and the entire upper abdomen. The stomach and esophagus can then be dealt with as a unit. It is so much easier to plan the extent of the resection and to control bleeding with this exposure than it is by reaching through the diaphragm. I have used the incision through the chest, delivering the stomach into the chest and had considerable difficulty separating the bare area of the stomach from the diaphragm. Also by splitting the diaphragm it can be sutured to the gastric side of the anastomosis, thus leaving the stomach in the abdomen. I have not encountered any postoperative complications from this incision. One patient in six complained of slight tenderness of the costal arch.

(Slide.) I also had an opportunity recently to approach the left adrenal gland through a thoracico-abdominal incision. The patient was a 12-year-old girl with Cushing's syndrome. She was a little over 4 feet tall and weighed 207 lbs. The urologist who had demonstrated the adrenal tumor by x-ray was at a loss to find a suitable approach. He was afraid of damaging the tumor if he used the kidney approach and the patient was too obese for an abdominal approach. By removing the eleventh rib and splitting the diaphragm we were able to expose the retroperitoneal space directly over the tumor. The tumor lay in the perirenal fat like an egg in a nest. This indicates that a transthoracic approach to lesions of the adrenal gland or upper pole of the kidney is practical.

DR. EARLE B. KAY, Cleveland, Ohio.—In answer to Dr. Sweet's discussion:

The esophagus was removed in three of the four intrathoracic esophagoplasties performed for undilatable, benign esophageal stricture. It was left in the fourth because the condition of the patient would not permit prolonging the operative procedure. It is felt that the esophagus in such cases should be removed because of known instances of later carcinomatous proliferation.

I agree with Dr. Sweet that the antethoracic jejunodermatoesophagoplasty is not as satisfactory as an intrathoracic procedure. However, I believe that there will continue to be instances in which it would be inadvisable to perform a reconstructive intrathoracic esophagoplasty.

TECHNICAL PROBLEMS IN THE SURGICAL TREATMENT OF CARCINOMA OF THE ESOPHAGUS AND UPPER STOMACH

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DURING the past ten years the surgical treatment of cancer of the esophagus and cardia has been extended in this country to such a degree that it is fair to state that the surgical approach to this problem has been accepted in most clinics as the only form of therapy so far developed that will at the same time give a low operative mortality and the longest period of postoperative survival. The road traveled in the development of this field of surgery has not been an easy one. There has been a constant probing for methods that would lead to earlier diagnosis, repeated changes in the development of the technical problems encountered during the operative procedures, and the gradual realization of the great importance of the patient's care during the postoperative period. All of these efforts have been expended with the purpose of attaining some form of standardization in the operative treatment of this disease. On the basis of experience with over 200 cases, I would like to summarize some of our experiences with the technical problems in the operative treatment of cancer of the esophagus and upper stomach.

In trying to cope with the high incidence of cardiovascular accidents occurring postoperatively, which was the subject of a paper presented two years ago before this association, we have increased the period of preoperative preparation and made free use of the services of our medical consultant. There seems to have been some decrease in the number of this variety of postoperative complications. During the preoperative period, careful attention is now being given to the prophylaxis of pulmonary complications. These preventive measures include, first, parenteral administration of penicillin and frequently of sulfadiazine, and second, the intratracheal insufflation of penicillin by nebulization. It is our impression that there has been a marked decrease in the number of postoperative pulmonary complications since this preoperative routine was adopted. We still emphasize the importance of dental prophylaxis during the preoperative period as an additional aid in the prevention of pulmonary infections. Mechanical cleansing of the esophagus itself by frequent irrigations of the organ through a Levin tube is another important adjuvant in the prophylaxis against postoperative infections.

There is no need to emphasize the importance of careful preoperative preparation in the type of patient under discussion. Dehydration, avitaminosis, disturbances of protein metabolism, and variations in the mineral components

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Read at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

of the body are frequent concomitants of the disease when the patient first presents himself. Various methods adopted to overcome these deficiencies are too well known to merit extended discussion at this time. However, it is important to emphasize that they should not be forgotten during the preoperative period.

Anesthesia.—The rapid advances in the field of anesthesiology have made continued progress in this field of surgery possible. We have gone through the various phases of anesthesia development in relationship to esophageal surgery and have finally come to the routine use of intratracheal anesthesia, which has now reached a high degree of efficiency. Anesthetists are now so well trained in the use of the intratracheal tube that there need be no hesitancy on the part of the surgeon to utilize this anesthetic modality in these frequently debilitated patients. It seems hardly necessary to emphasize that at the completion of the operation the anesthetist should routinely aspirate the bronchial tree to prevent occlusion of one or the other main bronchus postoperatively.

Midthoracic Carcinoma.—Since my original report of the high anastomosis for carcinoma of the middle third of the esophagus, enough experience has accumulated in other clinics as well as my own to justify the statement that the old Torek operation is now completely outmoded. It is now possible to bring the stomach to the apex of the chest and effect a direct anastomosis between the stump of the esophagus and the transplanted gastric organ. The important problem in this procedure is one of preservation of blood supply. It must be remembered that the only source of supply for the stump of the esophagus in this situation is through branches coming from the inferior thyroid artery. Therefore, every effort must be made to avoid trauma and extensive mobilization of the esophagus at the apex of the chest. With respect to the blood supply of the transplanted stomach, I originally suggested that the main left gastric artery should not be ligated. However, since that time experience has shown that it is feasible to ligate and divide the left main gastric artery as it arises from the coeliac axis. This maneuver alone permits extensive mobilization of the stomach. Should more mobilization be needed, the gastrocolic omentum may be divided in such a way as to preserve the gastroepiploic arch of vessels along this border of the stomach. If this is done with care, and this mobilization may be extended along the greater curvature almost two-thirds of the way to the pylorus, then the surgeon need have little fear of impairment of circulation in the proximal end when it is transplanted to the apex of the chest. We have had only one instance of necrosis of the gastric wall at the upper end of the transplanted stomach, and I am sure this was due to too extensive ablation of the blood supply along the greater curvature.

The approach that we have been using for this operation consists of a long incision in the sixth interspace with division of the sixth, fifth, fourth, seventh, and eighth ribs near the spine so as to form a T-shaped incision of the thoracic cage. The judicious use of a rib spreader gives sufficient exposure for both the thoracic and abdominal manipulations through the diaphragmatic incision.

We employ routine crushing of the phrenic nerve in order to render the left leaf of the diaphragm immobile during the operative procedure and postoperative period. The technical features of the anastomosis itself have been stressed frequently and need no extended discussion at this time. Suffice it to say that experience has shown that it is important to use interrupted sutures, preferably of the nonabsorbable variety, in order to prevent postoperative stricture at the site of the anastomosis. We have used a two-layer anastomosis of fine silk, the inner one approximating mucosa to mucosa, and the outer, muscularis of the esophagus to serosa of the stomach in the form of interrupted mattress stitches. Each suture must be placed accurately and tied without tension. To prevent disruption of this suture line, which should never be made under tension, we have telescoped the stomach over the suture line and anchored it to the esophagus above and to the mediastinal pleura on each side all the way down to the diaphragmatic opening. Before the anastomosis is completed it is advisable to aspirate the stomach completely so as to have a thoroughly flaccid organ at the end of the operation. The repair of the diaphragm around the transplanted stomach is an important item in this operation. Interrupted sutures should be placed close enough to prevent herniation of abdominal contents into the chest. Every possible opening must be obliterated and the closure should be thorough. I emphasize this point because we had the unfortunate experience of herniation of bowel into the chest in two patients, causing the death of the individual in each instance. In the first case, the ileocecal region was found herniated between rather widely placed sutures, and in the second, one of the silk sutures tore out of the diaphragm, thus creating an opening large enough to permit herniation of the transverse colon. This acted as a check-valve on the complete emptying of the stomach and the patient died of respiratory distress because of increasing distention of the stomach. The clinical picture was quite obscure and difficult to explain. The autopsy which we were fortunate to obtain told the whole story.

Carcinoma of the Lower Third of the Esophagus and Cardiac End of the Stomach.—The biopsy specimen obtained during esophagoscopy will indicate the origin of the tumor. Squamous cell tumors originate in the esophagus itself and adenocarcinomas originate in the upper end of the stomach or cardia. An adenocarcinoma may grow upward along the esophagus for quite a distance. However, I have never seen a squamous cell tumor of the esophagus grow downward along the gastric wall. Some years ago I stressed the importance of determining operability in all cases of adenocarcinoma of the cardia before subjecting the patient to a transthoracic incision. I therefore suggested a preliminary abdominal exploration through a left rectus incision. In the inoperable cases, closure of the incision terminated the operative manipulation. If the lesion was found operable, the abdominal wound was closed, the patient was turned on his side and a left transthoracic incision was then made. During the past two years I have made use of a combined abdominothoracic incision which has simplified the problem immeasurably. The development of this incision has been a natural one during the progress of this work and includes the best features of the various incisions of this type that have been described heretofore. In the early days of this century, surgeons attempted to reach the cardiac end of the stomach without entering the chest, and all sorts of osteoplastic flap incisions were utilized by Marwedel, Von Mikulicz, Hedlbom, Ohsawa, and others. Humphreys of New York called my attention to the many excellent features of this approach, and during the past two years I have utilized this incision almost routinely for carcinoma of the upper stomach and lower esoph-

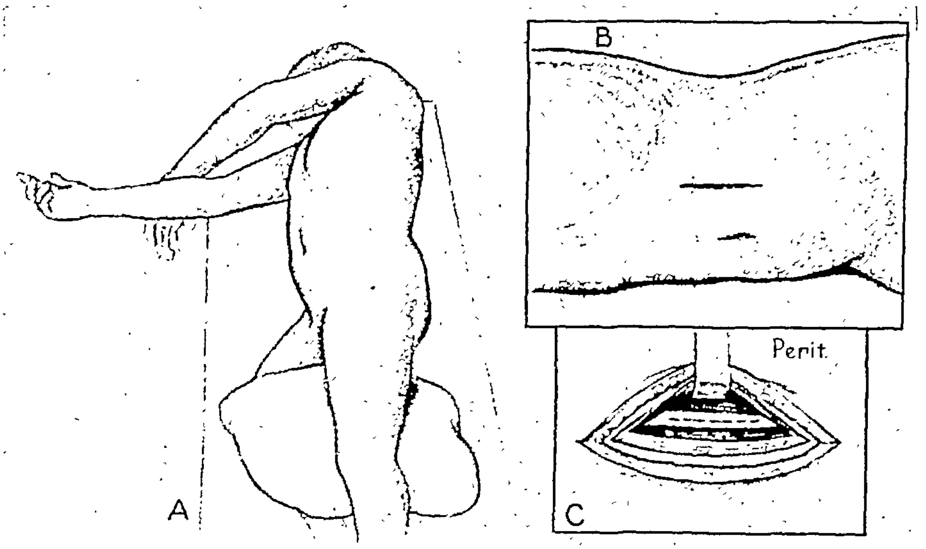

Fig. 1.—Position of patient on operating table. Beginning of incision at outer border of left rectus muscle. (Courtesy Surg., Gynec. & Obst.)

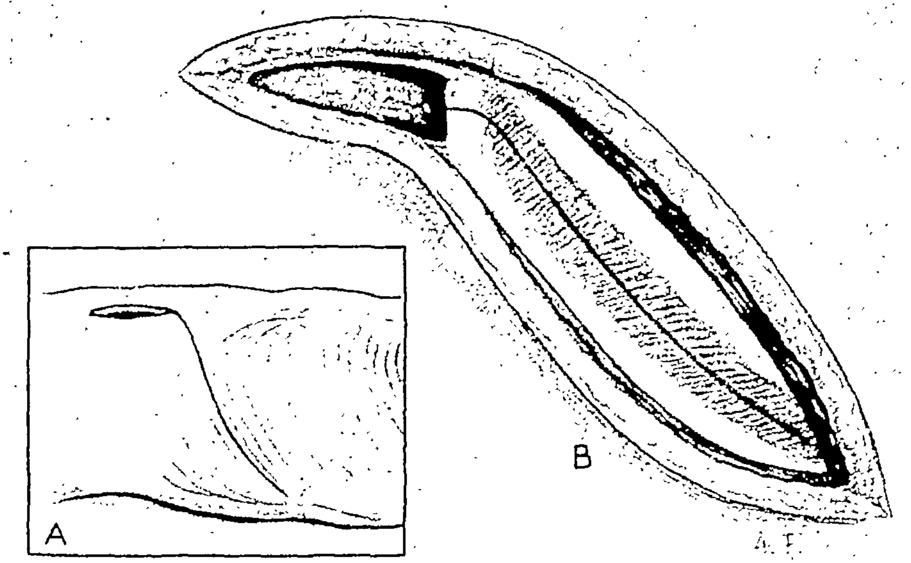

Fig. 2.—*A*, Continuation of abdominal portion of incision upward and outward into the eighth interspace. *B*, Division of the costal arch and intercostal musculature in the eighth interspace. (Courtesy Surg., Gynec. & Obst.)

agus. The incision affords a magnificent exposure of the upper abdomen and thoracic cavity. All operative manipulations can be done under direct vision with ease. More extensive resections can now be undertaken than was possible heretofore. It makes the operation of total gastrectomy a relatively easy procedure as compared with the usual operation done transabdominally. I have been impressed with the paucity of postoperative complications in these patients as compared with the previous group and also with the smoothness of convalescence.

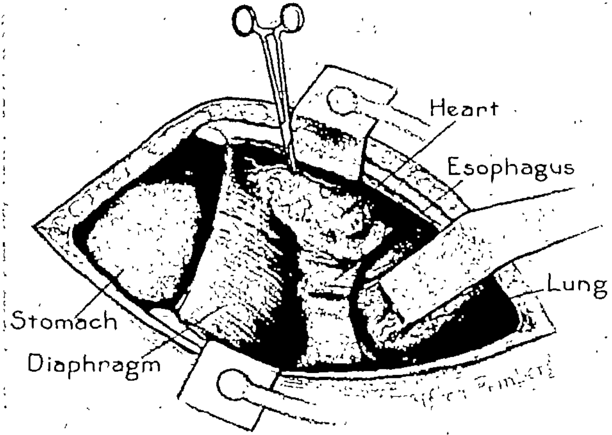

Fig. 3.—Radial incision of the diaphragm from the periphery to the esophageal opening. The phrenic nerve is pinched. (Courtesy Surg., Gynec. & Obst.)

The patient is placed almost in the exact lateral position on the edge of the operating table and an upper left rectus incision is made at the outer border of the muscle. (Fig. 1.) Exploration of the abdomen is then undertaken in order to determine operability. If an operable tumor is demonstrated, the incision is continued upward and outward in the eighth interspace. (Fig. 2.) The costal arch is divided at this point, and the incision is continued backward to the vertebral border of the scapula or further, if necessary. The chest is entered through the eighth interspace and the diaphragm is cut across in a radial direction to the esophageal hiatus. (Fig. 3.) A rib spreader is now inserted and opened widely, thus affording an extensive view of the upper half of the abdomen and lower thoracic cavity. (Fig. 4.) The esophagus, from the arch of the aorta downward, and the stomach may be mobilized with accurate division of the blood supply and complete removal of the associated lymph nodes. In squamous cell tumors of the lower esophagus it is not necessary to remove much of the upper stomach unless the paracardial lymph nodes are grossly involved. The latter is not an infrequent finding. More of the stomach must be ablated with carcinomas of the cardia in order to effectively remove the associated lymph nodes both along the lesser curvature and the paracardial region. It has been possible through this incision to effect block dissections of the upper stomach, tail of the pancreas, spleen, and the peripancreatic lymph nodes. This would have been a hazardous and extremely difficult procedure with the old transthoracic incision. (Fig. 5.) The repair of this wound is made in layers in exactly the same manner in which it was produced. The abdominal portion of the incision may be closed quickly with buried wire sutures of stainless steel. Before complete closure of the abdominal portion of the incision, jejunostomy may be done for postoperative feeding purposes. (Fig. 6.) The routine use of jejunostomy in this type of case has much to commend it. The thorax is drained by an underwater tube placed in a subjacent intercostal space. We have seen no harm done by the use of this tube and believe that it prevents accumulations of fluid in the chest which must otherwise be treated by repeated aspirations. Again I would like to stress the importance of accurate closure of the diaphragm around the transplanted stomach. This must be done in such a way as to prevent strangulation or diminution in the caliber of this organ.

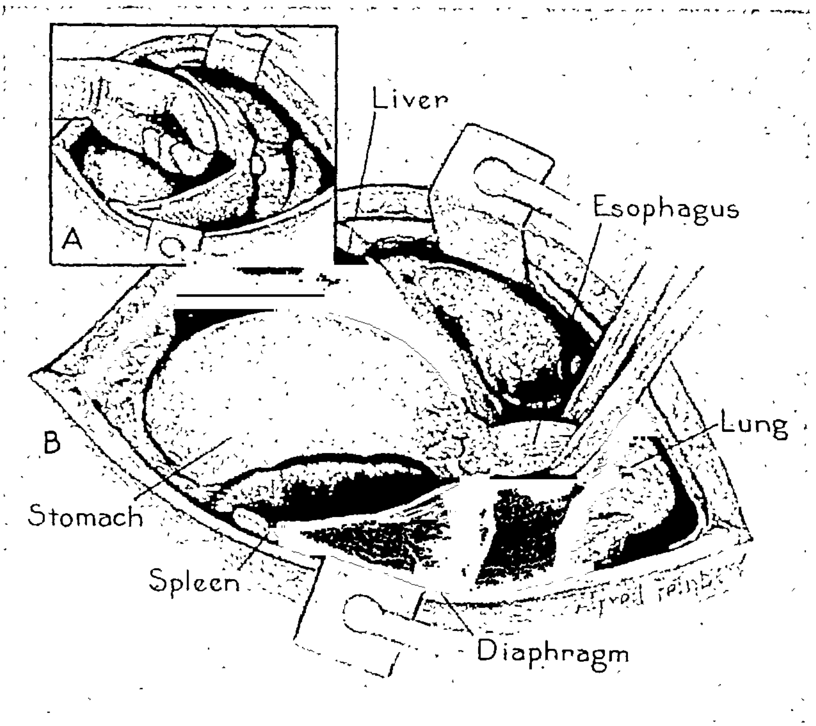

Fig. 4.—Mobilization of the lower esophagus and stomach. Lesion is located at the cardia. (Courtesy Surg., Gynec. & Obst.)

Total Gastrectomy.—I would like to mention briefly the desirability of using this combined abdominothoracic incision in the performance of total gastrectomy for carcinoma of the stomach. The technical details of the operation are tremendously simplified by the use of this incision. There is one other point that I believe deserves emphasis. In performing resections of the entire stomach and lower esophagus the surgeon is frequently confronted with the technical problem of a short jejunal loop. Sweet has attempted to overcome this defect by an exact ligation and division of the vascular arcades in the mesentery in order to obtain more mobilization. This has been a desirable advance and has simplified the problem somewhat. From the physiologic standpoint, considerable puddling takes place in the proximal or afferent loop of jejunum in spite of an enteroanastomosis, and this may produce undesirable digestive disturbances. To overcome the latter and at the same time to obtain added length to the jejunal loop, we have made use of the "Y" anastomosis of Roux during the past six months. A suitable loop of jejunum beyond the duodenojejunal junction is isolated and divided transversely. The arcades may be cut across with little fear of necrosis of the bowel ends. The distal end is inverted by two layers of silk. It is now approximated to the stump of the esophagus retrocolically, and an end-to-side anastomosis is accomplished in the usual manner. The proximal end of the divided jejunum is now anastomosed to the distal loop, end-to-side, about 8 to 10 inches below the esophago-

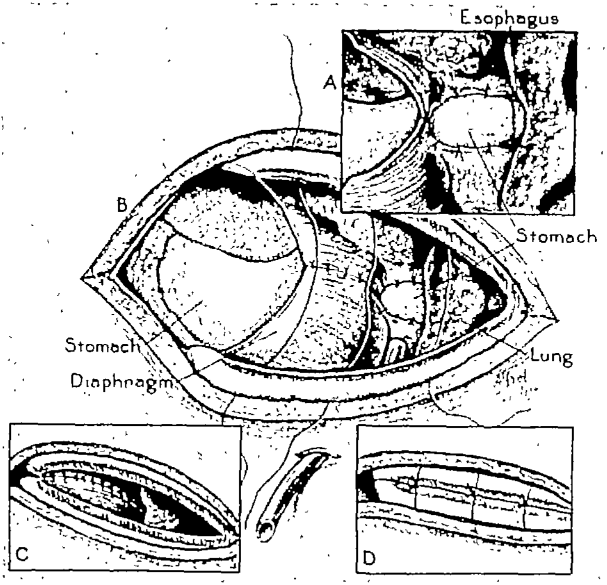

Fig. 5.—The anastomosis has been completed and the operative wound is being repaired. *A*, Fixation of mediastinal pleura and edge of diaphragm to transplanted stomach. *B*, Repair of diaphragm. *C*, Completed repair of diaphragm. *D*, Pericostal sutures inserted to close the thoracic portion of the wound. (Courtesy Surg., Gynec. & Obst.)

jejunal suture line. (Fig. 7.) The patients who have had this type of anastomosis have been singularly free of the upper abdominal digestive disturbances usually encountered in the ordinary loop anastomosis. It would seem that the proximal loop dilates and takes on the function of a reservoir for food and that puddling in the afferent loop is thereby prevented. I am suggesting this technical step as a possible improvement over the operation usually done for total gastrectomy.

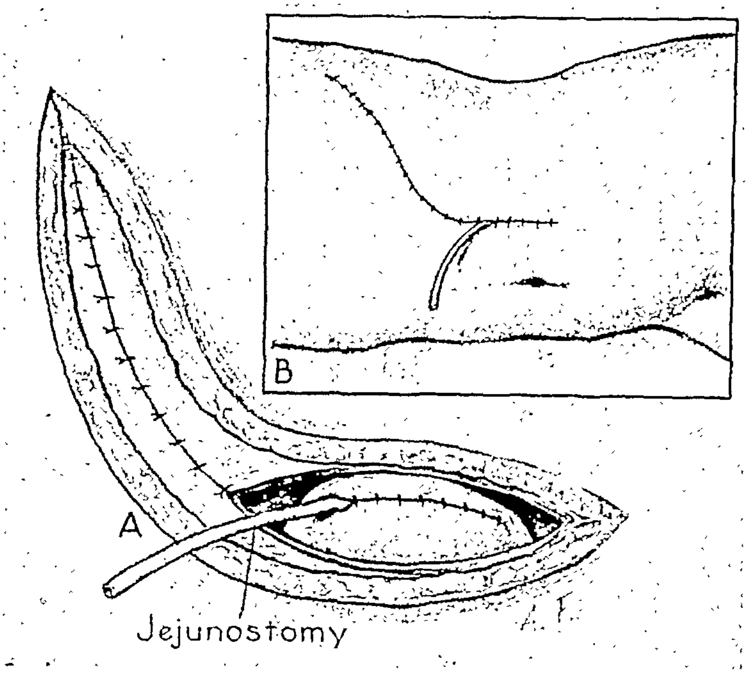

Fig. 6.—*A*, Further stage in the repair of the wound. Before closure of the abdominal portion, a jejunostomy is performed. *B*, Completed closure. (Courtesy Surg., Gynec. & Obst.)

Postoperative Care.—The postoperative care of the patients subjected to these operations is of great importance. Every patient is routinely placed in an oxygen tent immediately after operation. Thorough sedation during the first twelve to twenty-four hours is important because these patients do a lot of thrashing about during this time. Penicillin is administered parenterally. The chest is watched for accumulations of fluid and air. The most common site for trapped air is anteriorly below the clavicle on the left side. A tympanitic percussion note associated with dyspnea on the part of the patient calls for immediate aspiration. This should not be too long delayed. The clinical appraisal of the chest is probably more important than the routine bedside x-ray. Jejunostomy feedings may be started the day after operation. Of course, it is unnecessary to emphasize the importance of transfusions both during the operation and postoperative period. Swallowing is interdicted until the fourth day, when sips of water may be permitted. The quantity is rapidly increased until the seventh or eighth day, when junkets, custards, and gelatins are added to the diet. Thereafter the food intake may be stepped up so that the patient is able to enjoy practically a full diet by the end of the third week.

It has been astonishing to see these frequently debilitated patients go through these extensive resections with the postoperative chart simulating that of an interval appendectomy. The rapidly diminishing mortality rate of these operations has emphasized the importance of a combination of features.

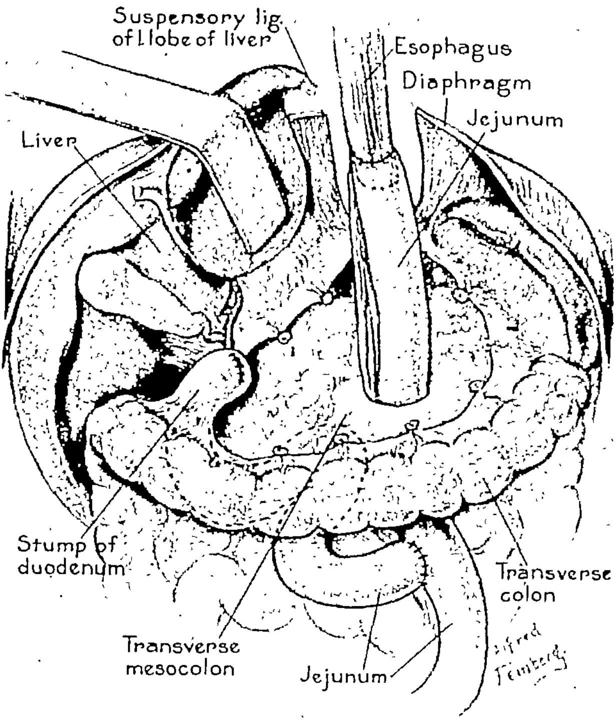

Fig. 7.—Diagrammatic representation of the completed operation of total gastrectomy performed through a combined abdominothoracic incision and showing the Y-anastomosis of the jejunum. About 18 inches beyond the duodenojejunal junction the jejunum and its mesentery has been divided. The distal end is brought through an opening in the mesocolon and sewed to the opened end of the esophageal stump in the usual manner. The proximal end of jejunum is implanted end-to-side into the distal portion below the mesocolon.

These may be enumerated as (1) careful preoperative preparation, (2) expertly administered anesthesia, (3) atraumatic surgery with minimal loss of blood, free exposure, and accurate suture methods, and (4) careful attention to the many details of the postoperative care. The increasing number of long survivals following these operations is indicative of the fact that we are becoming aware of the necessity for a thorough cancer operation which includes the wide removal of the cancer-bearing focus and all of its associated lymphatic tributaries. Until the exact cause of this disease becomes evident, our only hope in attaining an increasing percentage of cures is that patients will be referred at an earlier date for surgical treatment than has been the case heretofore.

DISCUSSION

DR. RUBIN M. LEWIS, Philadelphia, Pa.—Developments of all kinds have to come in their chronological order: most people who have had to do with upper abdominal surgery have found it preferable to use a one-incision approach. The incision we suggested is, perhaps, not entirely original for I am sure we picked up some ideas elsewhere. The impressing thing about the approach, and in contradistinction from the approach suggested by Dr. Garlock, is that we have recently become more and more impressed with the necessity for transverse incision, especially through the rectus muscle. I had occasion to examine one of these wounds about thirty days postoperatively, and found an excellent repair from a transverse rectus incision.

The danger of herniation through a transverse incision is a little less than through a vertical incision. The exposure that one gains by coming down either through the bed between the eighth and ninth ribs, or taking out the ninth rib, as we suggested, and going transversely across the rectus gives a much better exposure to the abdominal cavity and certainly gives an excellent exposure to the lower thorax, and perhaps a little more room than one can get with a vertical rectus incision; if the room is not adequate for a total gastrectomy, the right rectus incision may be used with this incisional approach.

DR. G. H. HUMPHREYS, New York, N. Y.—We have used the combined incision, such as Dr. Lewis described, starting with an obliquely transverse incision across the upper abdomen to determine operability. It is good for exploration, and through it a gastrostomy can be done if the lesion is inoperable. Then, by placing the patient in such a way that the table can be rotated, the patient is rotated and the incision carried upward into the chest, either excising the eighth rib or going through the eighth interspace and, if necessary, dividing the eighth and seventh ribs if the lesion runs as high as the pulmonary hilus. It has been very satisfactory.

I would also like to make one comment, on something Dr. Kay said about the desirability of doing an esophagogastric anastomosis rather than an esophagojejunal anastomosis. The results of our cases where only a very small stump of the stomach remained have been very disappointing. It seems to us that these patients have a great deal of difficulty with pyloric spasm, perhaps because there is not sufficient peristaltic activity in the remaining gastric stump. If the lesion is so large that most of the stomach has to be removed, it is probably better to do a total gastrectomy than a partial gastrectomy and try to pull a small gastric stump up into the chest.

DR. LOUIS R. DAVIDSON, New York, N. Y.—I should like to add my testimony relative to the advantages which are obtained with the use of the combined thoracoabdominal incision. Surgeons in New York have been stimulated by the work of both Dr. Garlock and Dr. Humphreys. In many instances the thoracic approach becomes confounding and the surgeon wonders whether he is doing the proper thing for the patient by merely opening the chest, incising the diaphragm, finding much to his consternation that he has an inoperable case, and of necessity backing out.

I was fortunate in witnessing Dr. Humphreys use his combined incision, and feel that he did not sufficiently emphasize the fact that he makes his abdominal incision first, and then after ascertaining that the lesion is operable, he continues in the fashion Dr. Garlock described.

I should like to add one point in regard to the incision. It is parallel with the eighth rib, as you saw, and runs down to the abdomen in the same direction. The exposure is magnificent.

In a case which was operated upon by me six weeks ago, we had adequate exposure, removing a little more than half the stomach, about three inches of the esophagus, the pancreas up to the origin of the coeliac axis, the spleen, and part of the left adrenal. The patient is about, walking. He had a slight empyema. This operation could never have been completed if it had not been for the thoracoabdominal incision, which not only permitted us to ascertain whether this case was operable but also gave very adequate exposure.

DR. JOHN H. GARLOCK, New York, N. Y.—There is very little to add. The smoothness of the postoperative course in these patients, and the relative absence of pain, is quite different for the convalescence of the patients who have had rib resections and rib splitting. I think the fact that we are not removing any ribs may be the explanation of that.

I agree with Dr. Humphreys that when you are left with a small stump of stomach it is much easier to take out the remainder than to anastomose a short prepyloric stump.

The pyloric spasm the patients have is due to the fact that one cuts both vagus nerves, and it is usually corrected by belladonna or atropine. This undesirable symptom may impede the patient's progress postoperatively, but usually it disappears spontaneously after a few months.

SUPRADIAPHRAGMATIC SECTION OF THE VAGUS NERVES TO THE STOMACH IN GASTROJEJUNAL ULCER

Lester R. Dragstedt, Ph.D., M.D. (by invitation), James S. Clarke, M.D.,
Paul V. Harper, Jr., M.D., Edward R. Woodward, M.D.,
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Chicago, Ill.

PREVIOUS reports from this clinic[1] have dealt with the technique of both supra- and subdiaphragmatic section of the vagus nerves to the stomach and with the results of this procedure on gastric secretion and motility in patients with peptic ulcer. The prompt relief of ulcer distress that invariably occurs and the objective evidence of the healing of the lesions have been described. Further work on these problems has been conducted in both the laboratory and the hospital, and it is the purpose of this communication to present the findings, some of which confirm and extend observations previously recorded. In addition, the results of gastric vagotomy in a series of intractable gastrojejunal ulcers will be described.

The theoretical basis for section of the vagus nerves to the stomach in the treatment of peptic ulcer is relatively simple and may be summarized in part as follows:

1. Present medical and surgical procedures that are effective in the treatment of peptic ulcer are designed to neutralize the acid gastric juice or to decrease its production. The first is usually attempted by frequent feeding and the administration of alkalies, and the second by subtotal gastrectomy or x-ray therapy to the stomach. To the extent that these measures accomplish their purpose, favorable results, in general, have been secured.

2. When an excessive continuous secretion of gastric juice is maintained in experimental animals, peptic ulcers appear in the stomach or duodenum, progress, and cause hemorrhage or perforation. This experiment of crucial importance was made by Wangensteen and his associates.[2] Excessive and long-continued gastric secretion was produced by implanting pellets of histamine and beeswax into the subcutaneous tissues.

3. An excessive continuous secretion of gastric juice occurs in most patients with peptic ulcer. The work of the older gastroenterologists demonstrated that patients with ulcers, on the whole, secrete a little more gastric juice in response to the stimulus of food, alcohol, or histamine than do normal individuals. The difference is, however, not very great, and is not sufficiently uniform to have much diagnostic value. It is in the activity of the empty stomach that the more striking contrast is seen. This is best determined at night when the patient is shielded from the sight and odor of food, and the stomach has been

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From the Department of Surgery, University of Chicago.

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carefully emptied and lavaged. A Levine tube is introduced through the nose into the stomach and constant suction maintained for twelve hours until the following morning. Under these conditions, an average of 250 to 400 c.c. of fluid is obtained from the stomach of normal adults and of patients with gall stones, regional enteritis, and miscellaneous diseases other than peptic ulcer. The free acidity of this aspirate ranges between 0 and 20 clinical units, indicating that the output of the parietal cells is small and that considerable dilution and buffering have occurred. Patients with duodenal ulcer, on the other hand, have uniformly been found to yield under identical conditions a night secretion of 600 to 2,500 c.c. with free acidity of 40 to 110 clinical units. If this is calculated in terms of hydrochloric acid content, it is apparent that the ulcer patient secretes from two to ten times as much acid as does the normal individual when there is no obvious stimulant to the gastric glands.

4. Section of the vagus nerves to the stomach reduces the secretion of gastric juice in normal animals and in patients with peptic ulcer to an even greater degree. The classic researches of Pavlov first demonstrated that the secretion of gastric juice which occurs in dogs in response to the sight, odor, or taste of food is abolished by cutting the vagus nerves. Precise data concerning the relative proportion of the total gastric secretion that is due to these reflexes over the vagus nerves has been difficult to obtain. It has long been known that secretion persists after complete vagotomy when food is present in the stomach or upper intestine. This is mediated by humoral, and possibly other factors, but its volume is uncertain. Orbeli[3] prepared an isolated Pavlov pouch in a dog and collected 23.8 c.c. of gastric juice in a seven-hour period following the ingestion of 100 Gm. of meat. The vagus nerves to the pouch were then divided, and when the experiment was repeated, the secretion fell to 4.3 c.c., or approximately one-fifth of the previous figure. Apparently, no attempt was made to collect the twenty-four hour secretion from the pouch before and after vagotomy. We are now studying this problem in our laboratory, and a detailed report will be made elsewhere. The entire stomach of the dog is isolated by transection at the cardia and pylorus.[4] The ends are closed, and a suitable cannula is inserted to collect the gastric juice. Care is taken not to interfere with the blood and vagus nerve supply to the isolated stomach. Continuity of the alimentary tract is re-established by anastomosing the lower end of the esophagus to the duodenum or to the side of the jejunum. With such a preparation, it is possible to collect all of the gastric secretion for periods of several weeks to a month or longer. One such animal secreted an average of 1,100 c.c. of gastric juice per twenty-four hours for a two-week period, with a free acidity of 116 clinical units. This fell to an average of 410 c.c. with a free acidity of 30 clinical units after division of the vagus nerves above the diaphragm. A second animal secreted an average of 500 to 800 c.c. with a free acidity of 33 to 90 clinical units before vagotomy, and this was reduced to 40 to 100 c.c. with a free acidity of 0 to 12 clinical units after the vagus section.

It is clear that in such a preparation neurogenic factors play a much greater role than humoral or other influences in determining the total volume of gastric juice produced in twenty-four hours and especially the output of acid. In the first case, the acid secreted was reduced by the vagotomy to one-tenth of the original amount, and in the second, to about one-sixtieth of that originally produced.

It is probable that a similar or even greater reduction in gastric secretion is produced by vagotomy in patients with peptic ulcer. It has been a standard procedure in this clinic to determine the night secretion in ulcer patients before and after section of the vagus nerves to the stomach. To date, such data have been obtained on 105 patients. The acid output of the stomach has been reduced by the operation to one seventh of the preoperative average and falls within the range of that found in normal individuals. This proves that the increased continuous secretion in ulcer is neurogenic in origin and is dependent upon a secretory hypertonus in the vagus nerves. Observations on the effect of vagotomy on the response of the gastric glands to the stimulus of food, histamine, and caffeine have also been made, largely by Mr. Harry Oberhelman, Jr. The concentration of acid resulting from histamine and caffeine has been unchanged, but the volume of secretion seems definitely reduced. It is exceedingly difficult to subject the gastric glands to the normal stimulus of food and then measure the volume of gastric juice produced. Data to date have been limited to the measurement of the concentration of free acid in fractional samples of gastric content removed at varying intervals after the meal. These will be reported elsewhere. They show that, whereas the well-known curve of increasing acidity of the gastric content was the rule before operation, after the vagotomy the gastric secretion was so reduced that free acid was not found, although fractional samples were studied for as long as five hours after ingestion of the meal.

The relief of ulcer distress following vagotomy has been so uniform, prompt, and complete as to suggest that the stomach has become anesthetic through removal of its sensory nerve supply. This is, however, not the case. Most of the patients subjected to subdiaphragmatic vagotomy have been given a spinal anesthetic. Under these conditions, mobilizing, pinching, tying, and cutting the vagus nerves have caused no sensation. A more crucial experiment was performed on two patients with duodenal ulcer who were experiencing severe distress when they entered the hospital. This distress was relieved temporarily by emptying the stomach but recurred in severe form when 200 c.c. of 0.5 per cent hydrochloric acid solution were instilled into the stomach (Palmer's acid test). Following vagotomy, both patients experienced immediate and complete relief. On the third day, while the patient's attention was diverted, the Wangensteen suction apparatus was disconnected and 200 c.c. of 0.5 per cent hydrochloric acid, warmed to body temperature, were injected slowly through the Levine tube into the stomach. Within ten minutes, severe epigastric pain similar in character to the previous distress appeared, and this was promptly relieved by withdrawing the injected acid. These findings suggest that the relief of pain produced by vagotomy is due chiefly to the marked reduction in the acidity of the gastric content. Doubtless, the reduction in the tonus and motility of the stomach also plays an important role, but the relief is certainly not due to anesthesia.

That section of the vagus nerves to the stomach causes the ulcers to heal is indicated by the persistent relief of symptoms in spite of cessation of all medication and dietary restrictions, gain in weight, and evidence of healing as determined by fluoroscopic study, i.e., disappearance of craters and lessening of deformities. In two cases of long-standing large gastric ulcers, these criteria have been supplemented by direct visualization with the gastroscope. Following vagotomy, the ulcers healed, and the mucosa in the ulcer area became nearly normal in appearance. Gastrojejunal ulcers are notoriously difficult to treat, spontaneous healing rarely occurs, and consequently, the response of these ulcers to vagus section is of more than usual interest. The following abbreviated protocols contain most of the relevant data in ten patients with gastrojejunal ulcers who have been treated to date in this clinic by gastric vagotomy.

I. O., a male aged 27, was first seen Jan. 12, 1943, complaining of epigastric pain coming on after meals and relieved by taking food or alkali. In 1936, a gastroenterostomy was performed for ulcer with obstruction. The distress was relieved temporarily but recurred in a few months. The patient frequently had diarrhea during exacerbations and occasionally had tarry stools. X-ray examination revealed a large crater in the jejunum near the gastroenterostomy with marked deformity and almost complete obstruction of the duodenal bulb. On Feb. 3, 1943, a subtotal gastrectomy was performed. The patient was relieved of symptoms for five months and gained much weight. He then began having severe epigastric pain and tarry stools. X-ray revealed a stoma ulcer. The patient was readmitted June 30, 1944, and a transthoracic vagotomy was performed. Following the vagotomy, the night pain disappeared. Cramplike epigastric pain appearing during the course of eating persisted, however, but vanished when the patient adopted the practice of eating slowly and small amounts at more frequent intervals. X-ray examination revealed complete healing of the ulcer. This probably represents a favorable response to vagotomy so far as healing of the ulcer is concerned, but persistence of symptoms due to the subtotal gastrectomy.

C. B., a male aged 42, was first admitted to this clinic Nov. 7, 1944. He complained of epigastric distress occurring several hours after meals and at night ever since he was 16 years old. The ingestion of food or milk always relieved the pain. In 1928, a posterior gastroenterostomy was performed for duodenal ulcer. Following this operation, his abdominal pain persisted and he developed diarrhea. He was operated upon again in 1930, at which time a gastrojejunocolic fistula was found. This was closed, and the gastroenterostomy taken down. The epigastric pain persisted, and in 1938 a subtotal gastrectomy was performed. This provided temporary relief, but early in 1944 symptoms recurred, and x-ray examination revealed a gastrojejunal ulcer. Medical management provided only partial and temporary relief, and he began to vomit. X-ray examination in this clinic, November 14, revealed a large jejunal ulcer. On November 18, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Recovery was uneventful. On Feb. 28, 1945, x-ray examination revealed a decrease in the size of the ulcer crater. The epigastric distress improved, and he began to eat very well, but was severely injured in an automobile accident in 1945. He was readmitted April 19, 1945, because of epigastric distress. The pain was not like the previous ulcer distress, but more a feeling of distention, coming on immediately after meals and not relieved by alkali, with no nausea or vomiting, but occasional sudden feelings of weakness. He left the hospital suddenly April 23, 1945, before studies were completed. Dr. Warren Cole has subsequently informed us that he performed a total gastrectomy on this patient because of obstructive symptoms. At this time, examination of the resected specimen showed that the ulcer had healed.

M. B., a male aged 56, was first seen in this clinic on June 20, 1942. Twelve years before, he began to have epigastric distress and consulted a physician, who made a diagnosis of duodenal ulcer. He was placed on medical management and did fairly well until ten days before entering the hospital, when he developed severe epigastric pain and had a massive hemorrhage. Subsequently, x-ray studies showed a duodenal ulcer with a large crater. X-ray therapy to the stomach was given and considerable improvement resulted. In November, 1942, he developed obstructive symptoms, and a posterior gastroenterostomy was performed. He was well for one year following this operation; then symptoms recurred, and x-ray examination revealed a gastrojejunal ulcer. Medical management failed to give relief. On Nov. 8, 1944, the gastroenterostomy was taken down, and on November 24, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Recovery was uneventful. When last seen in July, 1946, he reported that he had experienced no ulcer distress since the operation, had gained 40 lbs. in weight, and was eating a liberal diet and taking no medication. This represents a successful result to date, but is complicated by the taking down of the gastroenterostomy.

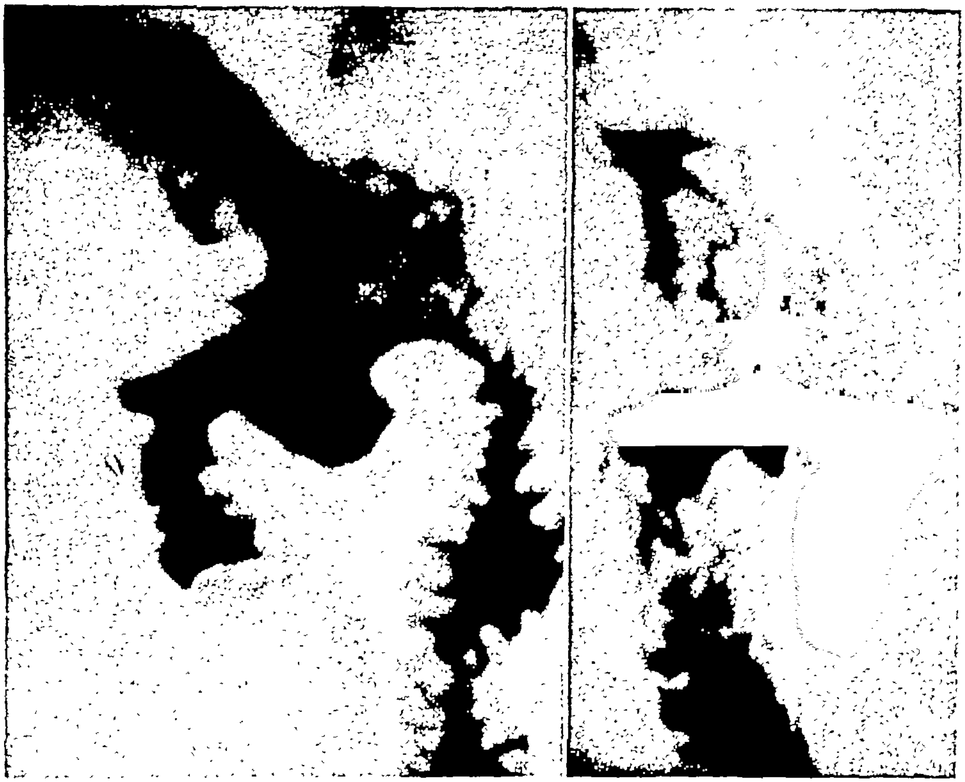

Fig. 1. Fig. 2.

Fig. 1.—Large gastrojejunal ulcer crater in patient G. P. appearing eleven months after subtotal gastrectomy.

Fig. 2.—Same patient as in Fig. 1, showing decrease in size of ulcer crater forty-nine days after section of vagus nerves to the stomach.

J. W., a male aged 60, was first seen in this clinic Sept. 15, 1942. He gave a history of epigastric distress for the previous twenty-five years. In 1936, a gastroenterostomy was performed. Epigastric distress recurred in 1940, and x-ray examination revealed a gastroenterostomy, and just above it, a large gastric ulcer. The ulcer was visualized with the gastroscope. X-ray therapy to the stomach was given, and on December 22, the ulcer disappeared and was not demonstrated on either x-ray or gastroscope examination. In August, 1943, symptoms recurred, and the ulcer reappeared at x-ray and gastroscopy. Strict management was instituted with some decrease in the size of the ulcer, but this increased again in the latter part of 1944. On Nov. 27, 1944, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Recovery was uneventful, but was complicated by persistent pain in the region of the incision. When last seen in September, 1946, he still complained of pain in the region of the chest incision. His ulcer distress was relieved from *the time of the operation, and no dietary restrictions were followed.* No medication was taken. This has been a successful result to date except for the persistence of pain in the chest.

A. W., a male aged 47, was first admitted to the clinic on Nov. 24, 1944. He complained of epigastric distress which had been present for the previous fifteen years. A diagnosis of duodenal ulcer had been made. Diet and powders controlled the pain fairly well during this period. Symptoms recurred, however, and two years before entering the hospital a gastroenterostomy was performed elsewhere. Following this operation he was well until four hours before admission, when he developed pain in his left side and began to vomit blood. The gastric hemorrhage was controlled, and on November 29, x-ray examination revealed a well-functioning gastroenterostomy with a gastrojejunal ulcer with a crater. On December 11, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Recovery from the operation was uneventful except for a slight pleural effusion on the left side which did not require tapping. Ulcer symptoms were entirely relieved, and x-ray examination on Jan. 9, 1945, failed to reveal the crater at the anastomosis. *When last seen in February, 1945, he was free from distress, eating a regular diet,* and taking no powders. The immediate response of this patient to vagotomy was very satisfactory, but the present status is unknown.

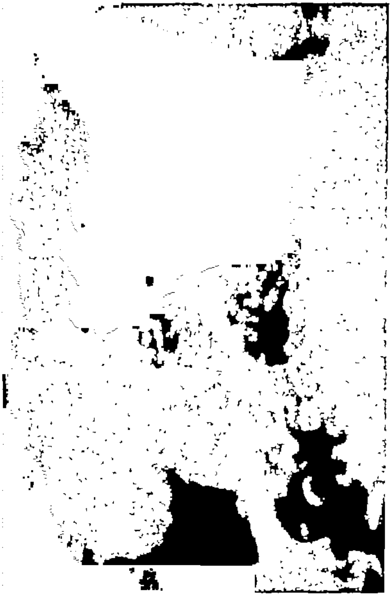

Fig. 3.—Same patient as in Fig. 1 showing complete disappearance of ulcer crater seventy-four *days after vagotomy.*

G. P., a male aged 48, was admitted to this clinic on March 2, 1944. Twenty years before, he first experienced abdominal pain accompanied by nausea and vomiting. A diagnosis of duodenal ulcer was made. Medical treatment proved unsuccessful, and in 1925 a gastroenterostomy was performed. This gave only temporary relief, and in 1939 x-ray studies revealed a gastrojejunal ulcer. He continued to have epigastric distress with occasional bouts of vomiting during the ensuing five years. When he entered the clinic, x-ray examination of the stomach March 8, 1944, revealed a large gastrojejunal ulcer. On April 3, the gastroenterostomy was taken down, and a subtotal gastrectomy was performed. Symptoms were entirely relieved until February, 1945, when the abdominal pain returned. X-ray examination on Feb. 14, revealed the reappearance of a very large gastrojejunal ulcer (Fig. 1). On March 12, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Immediate relief followed this operation, and he was able to eat a liberal diet without medication. Subsequent x-ray studies on April 30 (Fig. 2), showed decrease in the size of the crater, and on May 25 (Fig. 3), complete disappearance of the crater. When last seen in October, 1946, he was entirely well, eating an unrestricted diet without distress and with no medication. This represents a dramatically favorable response of an exceptionally large jejunal ulcer to vagotomy.

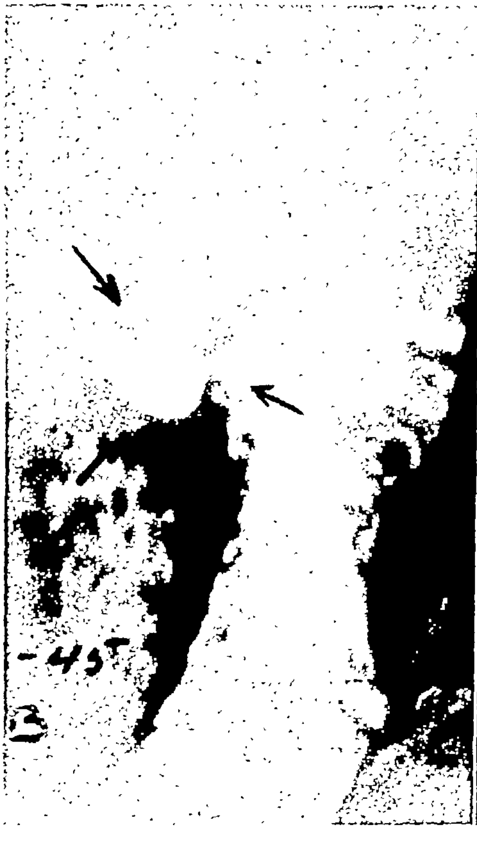

Fig. 4.

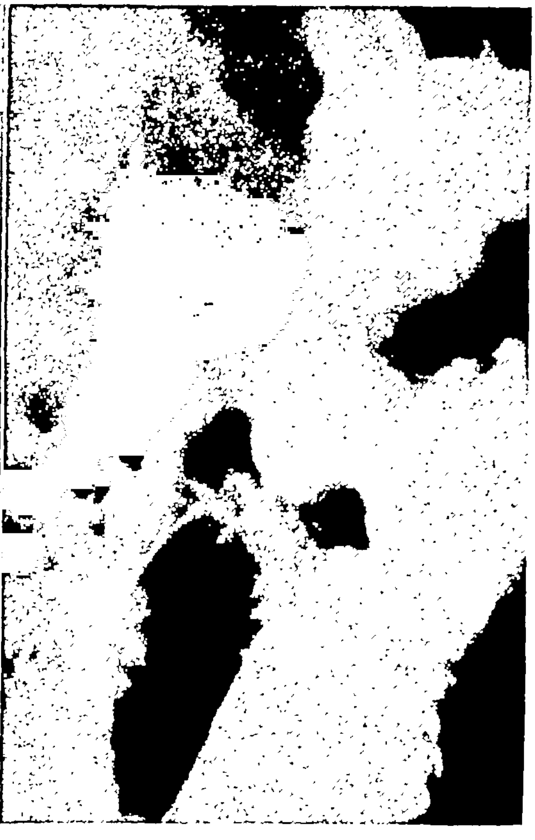

Fig. 5.

Fig. 4.—Large gastrojejunal ulcer crater in patient M. S. appearing six months after subtotal gastrectomy.

Fig. 5.—Same patient as in Fig. 4, showing decrease in size of ulcer crater twenty-four days after vagotomy.

M. S., a male aged 36, was first seen on Aug. 29, 1938. For the past two months he had been complaining of typical ulcer distress and x-ray examination revealed a duodenal ulcer with a crater. Symptoms were controlled for a time on medical management, but recurred in 1943, at which time a subtotal gastrectomy was performed. At operation, a chronic duodenal ulcer penetrating into the pancreas was found. Six months after the operation a recurrence of symptoms developed, and a gastrojejunal ulcer was demonstrated at x-ray. X-ray therapy to the stomach was then given with relief of symptoms, and there was apparent disappearance of the ulcer. The ulcer recurred in six months (Fig. 4) and pain became severe. On March 23, 1945, a transthoracic supradiaphragmatic section of the vagus nerves was performed. Recovery was uneventful, and the patient was entirely relieved of ulcer distress. X-ray examination on April 16, revealed definite decrease in the size of the ulcer crater (Fig. 5), and on May 9, complete disappearance of the crater (Fig. 6). When last seen in August, 1946, he was working regularly, eating an unrestricted diet without medication, and stated he had experienced no ulcer distress since the operation. This represents a strikingly favorable response of a large jejunal ulcer to vagotomy.

J. V., a male aged 50, was admitted Jan. 11, 1946, complaining of epigastric distress, coming on one to two hours after meals, of twenty-four years' duration. In 1923, the patient had a partial gastrectomy. Following this he was free of symptoms until four years ago when the ulcer distress recurred. The patient had tarry stools with each attack. For one year, pain had been severe and accompanied by vomiting. X-ray examination showed a large gastrojejunal ulcer (Fig. 7). On January 21, transthoracic vagotomy was performed by Dr. Hilger Jenkins. The postoperative course was uneventful. X-ray examination on April 26 showed complete healing of the stoma ulcer (Fig. 8). When last seen in October, 1946, he had gained 18 lb., was eating an unrestricted diet, and had no epigastric distress. No medication was taken at any time following operation.

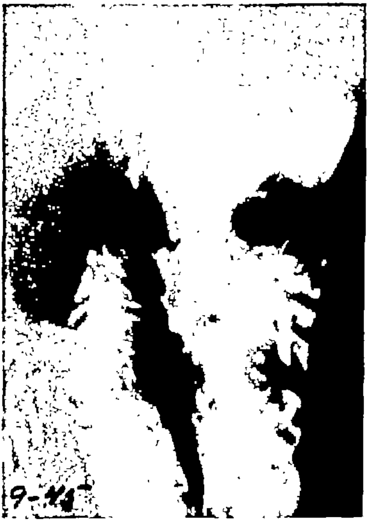

Fig. 6.—Same patient as in Fig. 4, showing complete disappearance of ulcer crater forty-seven days after vagotomy.

A. W., a male aged 59, was first seen on Jan. 10, 1946 with the complaint of burning epigastric pain of one and one-half years' duration. The distress came on about two hours after meals and was usually relieved for an hour or two by food, milk and cream, or alkali. He got along fairly well until three years ago, when intensive medical treatment for one year failed and a gastroenterostomy was done. The patient was symptom-free for about six months. The pain then recurred, has been more severe, and food and alkali have had less palliative effect. There was no history of perforation, stenosis, or hemorrhage. X-ray examination on Jan. 23, 1946, revealed a patent gastroenterostomy, at least one clean-cut jejunal ulcer, and probably a second shallow ulcer. Transthoracic vagotomy was performed February 11, and the postoperative course was entirely uneventful. When last seen in July, 1946, he had gained 10 lb., was working daily, and had no distress although he was eating an unrestricted diet and taking no medication.

M. S., a female aged 41, was admitted May 14, 1946, complaining of peptic ulcer of eighteen years' duration. In 1932, this patient had a gastroenterostomy performed for perforating duodenal ulcer. She had had symptoms for seven years previously. She was entirely well for five or six years after this, but then began to notice periodic gnawing epigastric pain coming on one and one-half to two hours after meals and relieved by milk. Each spring and occasionally in the fall, she would have an episode lasting five to eight days of vomiting, severe headache, and tarry stools. The patient was seen here in the medical clinic Oct. 19, 1943, at which time x-ray disclosed a moderate stenosis of the gastroenterostomy stoma with a definite crater. On medical treatment, including x-ray therapy, the crater disappeared and the patient felt well until eight months before admission. She then began having epigastric distress, and six weeks before admission had a massive hemorrhage.

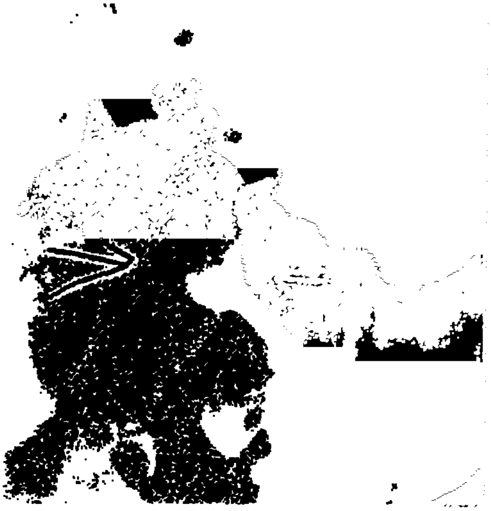

Fig. 7.

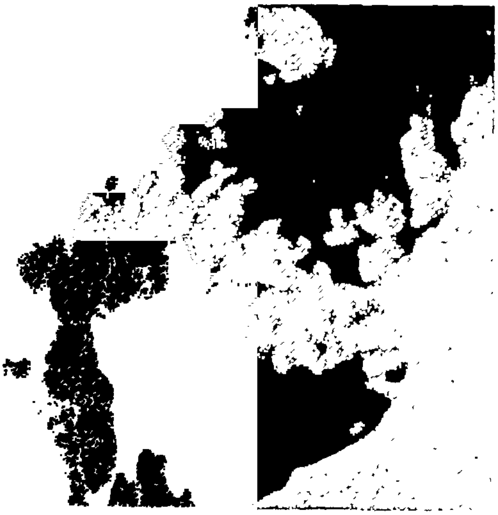

Fig. 8.

Fig. 7.—Gastrojejunal ulcer crater in patient J. V. appearing four years after partial gastrectomy.

Fig. 8.—Same patient as in Fig. 7, showing complete disappearance of ulcer crater ninety-five days after vagotomy.

The patient's father has had a subtotal gastrectomy for peptic ulcer, and three brothers all have peptic ulcer. The patient had chorea at the age of 9, and now has evidence of mitral insufficiency and stenosis without history of heart failure. Preoperative x-rays demonstrated a stomal ulcer in October, 1945, with considerable narrowing. In April, 1946, the crater could not be visualized, but the stoma was almost completely obstructed. The transthoracic vagotomy was performed on May 17, 1946. Postoperative recovery was uneventful except for three days of unexplained lethargy and semistupor. Patient was discharged on the tenth postoperative day. One month after operation, the patient had no ulcer pain or distress, no diarrhea, no dietary restrictions, and no medication. She still has occasional pain in her incision. X-rays show the gastroenterostomy to be functioning normally without evidence of crater.

DISCUSSION

The dramatic healing of large gastrojejunal ulcers as a direct result of section of the vagus nerve supply to the stomach is perhaps the best evidence of the therapeutic effectiveness of this procedure. Spontaneous healing of these lesions rarely occurs, and they are often refractory to the most rigid dietary and medical management. In addition to pain and distress resulting from the presence of the ulcer itself, many of these patients suffer from obstructive symptoms due to deformity of the jejunum and secondary inflammatory reaction of the neighboring tissues. It is important to recognize this complication since, of course, the obstruction is often not relieved by the vagotomy even though the ulcer heals and gastric retention may become even more marked as a result of the decrease in tonus and motility of the stomach. Our second case falls in this category, and the patient was dissatisfied with the operation because of the persistence of obstructive symptoms. Recurrence of ulcer following subtotal gastrectomy is occasionally complicated by symptoms arising as a result of the absence of a large part of the stomach or from distention of the portion of jejunum employed for the anastomosis. These factors are probably illustrated by our first case. This patient was a sailor and was accustomed to bolting his food. Vagotomy relieved his ulcer pain and brought about disappearance of his ulcer crater, but he continued to experience epigastric distention and cramps during meals. This was relieved when he adopted the practice of eating small amounts of food at more frequent intervals.

Complete division of the vagus nerve supply to the stomach may be accomplished by the thoracic or abdominal operation. The better exposure of the lower esophagus secured when the left chest is opened makes it somewhat easier to isolate the vagi and to make certain all gastric fibers are divided. The use of this approach in gastrojejunal ulcers following subtotal gastrectomy or other multiple abdominal operations has the additional advantage of avoiding the sometimes troublesome adhesions commonly present. An undeniable disadvantage results from inability to examine the lesion directly and to deal with obstruction, should it be found. In two recent gastrojejunal ulcers treated by us and not included in this report, vagotomy was accomplished easily by the abdominal operation. Further experience is necessary before a final decision regarding the best approach for vagus section in these lesions can be reached. Individual factors such as body contour and amount of fat also bear on the problem.

The finding that gastrojejunal ulcers heal following vagotomy is of interest in connection with the employment of gastroenterostomy in ulcer patients. In many clinics, this operation has been entirely abandoned or used only in old people with high-grade cicatricial pyloric stenosis and low acid concentration in the gastric secretion. For the most part, the high incidence of gastrojejunal ulcer has been responsible for this disfavor. To date, we have employed transabdominal vagotomy plus gastroenterostomy in fifty patients with duodenal ulcer. Many of these had pyloric obstruction as indicated by symptoms and fluoroscopic examination. The results so far have been excellent, and no secondary stomal ulcers have developed. While the period of observation is still too short for this data to be of significance, it seems highly probable that since vagotomy permits an already established stomal ulcer to heal, a new lesion of this type should not develop in a normal mucosa.

CONCLUSIONS

1. Ten patients with gastrojejunal ulcers following gastroenterostomy or partial gastrectomy have been treated by supradiaphragmatic section of the vagus nerves to the stomach. Healing of these lesions has been secured, as evidenced by relief of distress and by fluoroscopic demonstration of disappearance of large ulcer craters.

2. The relief of ulcer distress uniformly produced by vagotomy is not due to anesthesia of the stomach. This is indicated by the immediate reappearance of typical pain when acid is instilled into the stomach during the first few days after operation.

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DISCUSSION

DR. D. T. SMITH, Durham, N. C.—One of our surgeons at Duke, Dr. Keith Grimson, has performed this operation of Dr. Dragstedt's on about thirty patients in the last two years. In each case the approach was the supradiaphragmatic.

We have a whole team working on the problem, which requires the cooperation of roentgenologist, gastroenterologist, and neurologist. In all cases the pain has been relieved immediately and there has been no recurrence of bleeding in any of these patients. All patients have good appetite and are free of pain, but many overindulge in food. Some of them have developed large dilated stomachs and they have learned to massage manually to force the excess food through the pylorus.

DR. FRANK B. BERRY, New York, N. Y.—I should like to ask Dr. Dragstedt one question. That is the question of pylorospasm, as an after-effect of vagectomy.

Some of the neurosurgeons have told me that the operation is impracticable because of this complication, and pyloroplasty will necessarily follow. Inasmuch as Dr. Dragstedt has been performing this operation for three years, I would like to know his late results.

DR. LESTER R. DRAGSTEDT, Chicago, Ill.—With regard to the question of pyloric spasm following vagus section, I think it is true that a long-continued increase in the tonus of the pylorus is produced by vagotomy, but if there is not cicatricial obstruction of the pylorus, it does not seem to cause subsequent difficulty. In the majority of our patients ulcers were present in the duodenum. There was considerable deformity of the bulb and spasm, but following transthoracic vagus section, these patients, with the exception of two, have been relieved and eat large amounts of food. They have gained a great deal of weight and have had no subsequent gastric distress.

One of the patients went out on a picnic, ate a great amount of food and watermelon three months after the operation, and came to the hospital with an acute dilatation of the stomach. We decompressed him, operated upon him, and found a fibrous stenosis at the pylorus. The lumen had been decreased. We did a gastroenterostomy and subsequently he has remained well.

One problem for which I have no adequate answer is the degree of pyloric obstruction that will require gastroenterostomy in addition to the vagus section. It is difficult to determine. At the present time I follow this rule. If the patient gives a clinical story of retention with repeated vomiting, a gastroenterostomy is performed.

AN ANNOUNCEMENT OF OPPORTUNITIES FOR THORACIC SURGEONS IN VETERANS' ADMINISTRATION HOSPITALS

BRIAN BLADES, M.D.
WASHINGTON, D. C.

GENERAL HAWLEY asked me to appear before this Association to emphasize the need for thoracic surgeons in Veterans' Administration Hospitals.

In order to attract desirable surgeons, three factors have been considered: (1) provisions for large enough surgical services to furnish an adequate volume of clinical material and opportunities for clinical research; (2) adequate pay; and (3) freedom from administrative duties which will interfere with professional work.

It is possible to state without qualification that opportunities for clinical research in thoracic surgery are available in many veterans' hospitals, particularly in the field of tuberculosis.

Financial remuneration is based on the surgeon's previous training and ability. Men certified by their respective specialty boards receive 25 per cent more base pay. This is, of course, an obvious attempt to attract well-trained surgeons.

The Department of Medicine and Surgery in the Veterans Administration is no longer under Civil Service. Every attempt is being made to eliminate nonprofessional administrative duties which would obstruct a pleasant and useful professional career.

There has been the inevitable confusion during the period of reorganization. Many applications for positions in Veterans Administration hospitals have not been acknowledged promptly. The plan of organization is not generally understood and in some instances applicants have obtained conflicting opinions. For this reason I should like to describe very briefly the present organization.

In the Department of Medicine and Surgery headed by the Chief Medical Director, General Hawley, all of the principal medical and surgical specialties are represented at the central office by directors in the particular field. Dr. Elliott C. Cutler is the Chief of Professional Services.

One of the first objectives in the development of veterans hospitals is to decentralize the organization. Accordingly, branch directors in the various specialties have been appointed for the thirteen branch areas into which the country is divided (Figs. 1 and 2). The appointment of the branch director in thoracic surgery in Area No. 11 has not been made but will be announced soon.

In cities where the geographic location of medical schools make it feasible to have the school or schools assume the responsibility of staffing hospitals, the Dean's Committee Plan is in operation if the school is willing to cooperate.

Read at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

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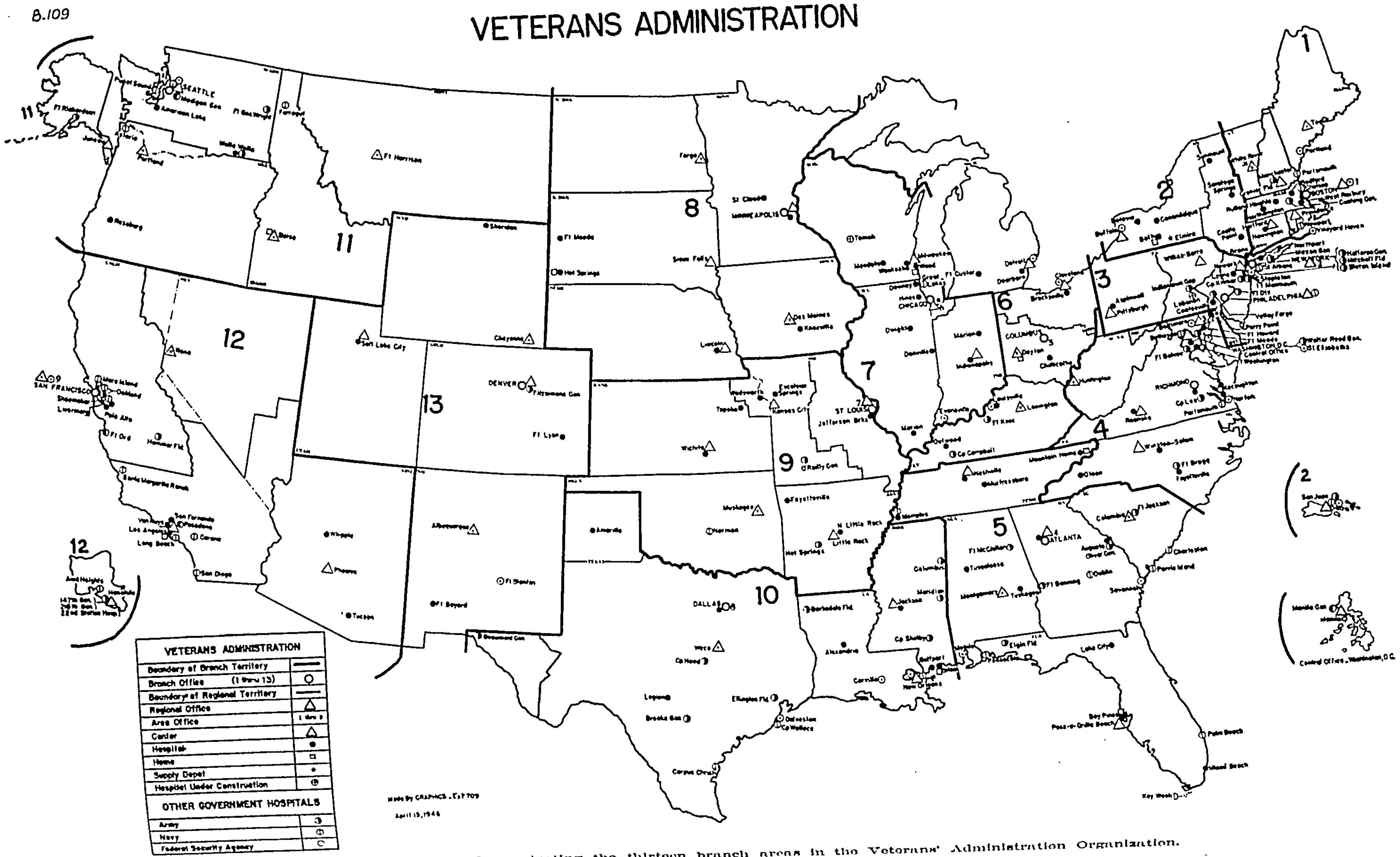

FIG. 1.—Map demonstrating the thirteen branch areas in the Veterans' Administration Organization.

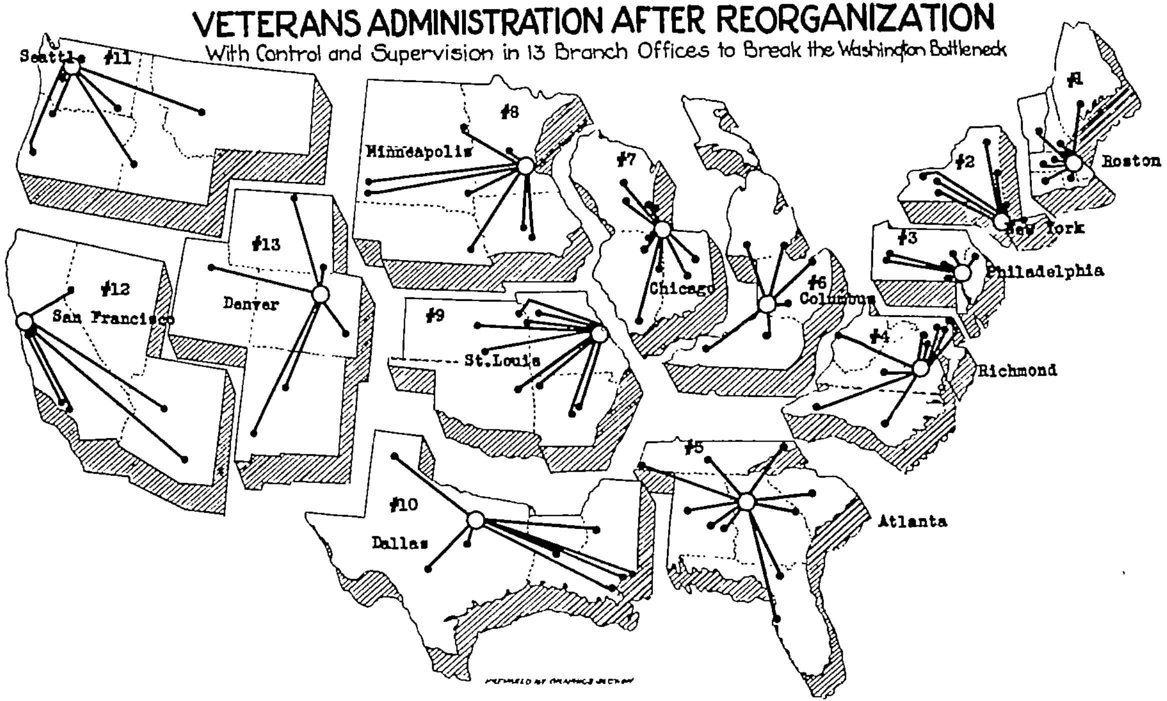

Fig. 2.—Map showing the locations of the branch area and regional offices of the Veterans' Administration.

TABLE I. BRANCH DIRECTORS IN THORACIC SURGERY

NAME AND ADDRESS	AREA	LOCATION OF BRANCH OFFICE
Dr. Gustaf Lindskog Yale University Medical School New Haven, Conn.	1	Boston
Dr. Warriner Woodruff 8 Church Street Saranac Lake, N. Y.	2	New York
Dr. Edward M. Kent University Club Pittsburgh, Pa.	3	Philadelphia
Dr. Everett Drash Univ. of Virginia Richmond, Va.	4	Richmond
Dr. Edward F. Parker, Jr. 70 Hasell Street Charleston, S. C.	5	Atlanta
Dr. William M. Tuttle 307 David Whitney Bldg. Detroit, Mich.	6	Columbus
Dr. Joseph Gale Univ. of Wisconsin Madison, Wis.	7	Chicago
Dr. O. T. Clagett Mayo Clinic Rochester, Minn.	8	Minneapolis
Dr. Thomas Buford Dept. of Surgery, Barnes Hosp. 600 South Kingshighway St. Louis 10, Missouri	9	St. Louis
Dr. Michael DeBakey Dept. of Surgery Tulane Medical School New Orleans, Louisiana	10	Dallas
Dr. Paul Samson 2938 McClure Oakland, California	12	San Francisco
Dr. John B. Grow Fitzsimons Gen. Hosp. Denver, Colorado	13	Denver

The Dean's Committee has authority to nominate for appointment residents, attending surgeons, and senior consultants, and is charged with the responsibility of the educational program in the hospital. It is necessary, therefore, to make application directly to the Dean's Committee for positions in hospitals supervised by the medical schools. The Research and Education Service, directed by Dr. Paul B. Magnuson, is responsible for the Dean's Committee Program.

In hospitals not supervised by medical schools, applications for positions may be made directly to the branch medical director, central office, or to the manager of the hospital.

There is not sufficient time to discuss in detail the various available positions and pay schedules. Complete information concerning opportunities in thoracic surgery in Veterans Administration hospitals may be obtained from the branch director or from the central office.

DIAGNOSIS OF TETRALOGY OF FALLOT AND INDICATIONS FOR OPERATION

HELEN B. TAUSSIG, M.D. (BY INVITATION)[*]
BALTIMORE, MD.

THE four features which characterize the tetralogy of Fallot are pulmonary stenosis or atresia combined with dextroposition of the aorta, a high ventricular septal defect, and right ventricular hypertrophy.

The characteristic clinical findings are cyanosis and clubbing, a heart of normal size, usually a basal systolic murmur, and a pure second sound. The electrocardiogram shows a right axis deviation. The x-ray shows an absence of the normal fullness of the pulmonary conus and clear lung fields. Fluoroscopy shows no pulsations at the hilar of the lungs.

There are many variables. The systolic murmur may be quite loud or entirely absent. The second sound may be diminished or accentuated; it is never reduplicated. Cyanosis may be marked or slight, depending upon the degree of the oxygen unsaturation and the height of the red blood count. The oxygen saturation of arterial blood is also subject to great variation (from 88 to 10 per cent). It varies from individual to individual depending upon the severity of the pulmonary stenosis, and also in the same individual from day to day. The exercise tolerance of the individual shows a corresponding variation: some persons can walk a mile, some not at all. Operation is indicated for those who are severely handicapped.

The operation is designed to increase the circulation to the lungs. It consists of an anastomosis between the proximal end of one of the systemic vessels to the side of one of the main pulmonary vessels. The operation is indicated only in cases in which the primary difficulty is lack of circulation to the lungs. It is possible only provided there is a pulmonary artery to which to anastomose the systemic artery and provided the pressure in the systemic artery is higher than in the pulmonary artery, and finally, that the size and structure of the heart is such that it can adjust to the altered circulation. Fluoroscopy aids in the determination of the size and structure of the heart. The heart should not be enlarged. There is no fullness of the pulmonary conus and no pulsations in the lung fields. Usually there is an absence of hilar shadows. However, in patients with extensive collateral circulation these shadows may become pronounced but are composed of an aggregate of fine shadows and never show expansile pulsations.

The diagnosis of a right or left aortic arch is of importance because the position of the aorta determines the side upon which operation is performed.

Summary of paper read at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30 and 31, 1946.

[*] Abstracted at Johns Hopkins Hospital, Baltimore, Md.

THE TECHNIQUE OF CREATION OF AN ARTIFICIAL DUCTUS ARTERIOSUS IN THE TREATMENT OF PULMONIC STENOSIS

ALFRED BLALOCK, M.D.
BALTIMORE, MD.

THIS paper consists of a discussion of some of the important technical points in the construction of an artificial ductus arteriosus such as Dr. Taussig and I have employed in the treatment of congenital pulmonary stenosis.[1-4] These comments, which are based upon 144 operations* upon patients who were thought to have an inadequate flow of blood to the lungs, are presented with the hope that others may be spared some of the mistakes.

Cyanosis and Disability.—Most of our patients have shown cyanosis even when at rest. It is proper when a new operative procedure is undertaken that one should choose the patients who have the least to lose in case the operation should result fatally. A number of our patients have required more or less constant exposure to a high concentration of oxygen prior to the operation. It is only natural that the mortality rate in such patients will be higher than in those who are not so ill. A few of our patients have had very little visible cyanosis when at rest but have been unable to tolerate exercise. It should be borne in mind that it is mainly the incapacity and not the peculiar color for which the operation is performed. If there is a history of intolerance to exercise, the oxygen saturation of the arterial blood should be determined immediately after limited exertion.

Age of Patient.—The patients in this series have ranged in age from 2 months to 26 years. The youngest patient on whom the operation has been performed successfully was 5 months of age and the oldest was 26 years. The ideal age is between these extremes and probably lies between 3 and 7 years. The upper and lower age limits in which the operation may result successfully have not been determined; patients under 18 months of age should not be operated upon if it is thought that they will survive to a greater age. There are several reasons for this limitation: (1) One can be less certain as to the correctness of the diagnosis in infants; (2) the mortality rate accompanying operation is higher in this age group; and (3) it is frequently necessary in infants to utilize the innominate or the carotid artery rather than the subclavian, and this adds to the danger of the operation.

Anesthesia.—General anesthesia has been induced by the use of inhalation agents in all of the patients. Dr. Lamont and Dr. Harmel have employed cyclopropane with a high concentration of oxygen in most of the operations. Ether with oxygen has been used in a few, and a combination of ether, cyclopropane, and oxygen in others. It has been very gratifying to note that the color of most patients improved when they were anesthetized. It has not

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From the Department of Surgery of the Johns Hopkins University and Hospital.

Read at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30 and 31, 1946.

*This number has now reached 450 and a few alterations in technique have been made.

been determined whether this change is due to the fact that the patient becomes quiet and relaxed or whether it is due to the high concentration of oxygen. The former would appear to be more likely.

Intravenous Fluids.—A needle is placed in a vein of the ankle prior to the beginning of each operation, and salt solution, or glucose solution, or both are administered slowly. Both blood plasma and whole blood are available. The latter is given only if the loss of blood is believed to be excessive. Whenever the operative procedure is a proonged one, blood plasma is administered.

Site of Incision.—The patient is placed on his back; the side of the chest on which the incision is to be made is slightly elevated and the incision extends from the lateral margin of the sternum to the midaxillary line. Usually the incision is made beneath the breast. The pleural cavity is entered through the third interspace and the third costal cartilage is divided. A rib spreader is introduced. When working on the left side, one has to take care that the retractor does not press against the heart and interfere with its action.

The present plan is that the incision is made on the side opposite that on which the aorta descends. The reason for this is that it is desirable to gain access to the innominate and its branches, and this vessel lies on the right when the aorta descends on the left and on the left when the aorta descends on the right. When the innominate is exposed, one may usually choose the innominate or its subclavian or carotid branches for anastomotic purposes, depending upon the size of vessel desired. Even more important, since the subclavian artery is the vessel usually chosen, is the fact that the subclavian branch of the innominate makes a much better angle with its parent vessel following the performance of an anastomosis than does the subclavian which arises from the aorta. The side on which the aorta descends can be determined by the method of Bedford and Parkinson.[5] The aorta has descended on the right in 38 of our operative cases, and in these the incision has been made on the left. Dr. Taussig has determined correctly the position of the aorta prior to the operation in all cases.

Exposure of Pulmonary Artery.—After the pleural cavity has been opened and a rib spreader has been introduced, the pulmonary artery is exposed. This step is facilitated when working on the right side by preliminary ligation and devision of the azygos vein. Traction on the ligature on the proximal end of the azygos vein aids in exposure of the pulmonary artery and the mediastinal contents. By the use of sharp and blunt dissection, the right or left pulmonary artery is dissected free of the surrounding tissues and a piece of heavy braided silk is placed around it. The exposure is usually more difficult on the right than on the left. It is necessary to free an adequate length of the vessel and there is no hesitation in opening the pericardium. In the majority of the cases, however, the pericardial cavity has not been entered. After the braided silk has been placed around the right or left pulmonary artery, the main branches of the vessel are exposed. In order to gain added length for the purposes of the anastomosis, pieces of braided silk around these branches replace that around the larger vessel.

After exposure of an adequate length of the pulmonary artery, the pressure within the vessel is usually measured by the use of a needle and a water manometer. If the vessel is small and the pressure within it is obviously low, this test is omitted for fear of damaging the artery. A large pulmonary artery with moderate pulsations does not mean necessarily that the pressure is high or that the flow is great or that the patient will not be helped by an anastomosis. The pulmonary arterial pressure was measured in 68 cases. In 56 of these it was less than 250 mm. of water. The most frequent reading was between 150 and 200 mm. If the pressure is greater than 350 mm., it is my belief that the anastomosis should not be performed unless other evidence points strongly toward the presence of a significant reduction in pulmonary blood flow.

Abnormalities of the pulmonary artery have presented the greatest difficulties that have been encountered. In two patients, one of whom was operated upon, there was a pulmonary artery to the right lung but none to the left. This is shown diagramatically in Fig. 1. The left lung received most of its blood through a branch of the innominate artery. In two patients who died, the right pulmonary artery was small and was posterior and inferior to the superior pulmonary vein and was not found at operation. The pulmonary artery was too small for a satisfactory end-to-side or end-to-end anastomosis in four patients. In at least thirteen patients the right pulmonary artery divided very shortly after its origin from the main vessel. In two instances I mistook the vessel to the right upper lobe for the right pulmonary artery. This mistake will probably be made frequently unless one is aware of the importance of identifying the right main pulmonary artery. It may be necessary to open the pericardium in order to expose it. If after careful freeing of the vessel it appears to be too short for an end-to-side anastomosis, it is advisable to ligate it proximally, to divide it, and to do an end-to-end anastomosis. In freeing the very short and smaller vessels the walls may be so weakened by division of the supporting tissues that it will be difficult if not impossible to perform a satisfactory end-to-side anastomosis.

In patients in whom the pulmonary artery is in an abnormal position or is absent, the superior pulmonary vein may be mistaken for the artery. I have had this unfortunate experience in two cases, although in both instances it was realized that the course of the vessel was that of a vein. Pulmonary edema and death resulted following the making of an anastomosis. If in doubt as to the nature of the vessel one should open the pericardium, but even then it may be difficult to be certain.

If it is decided after freeing the right or left pulmonary artery that the diagnosis is correct and that the structures are suitable for an anastomosis, the systemic artery that is to be used is exposed.

Exposure of Systemic Vessel.—The mediastinal pleura between the vagus and phrenic nerves is incised. When working on the right side, one should divide the mediastinal structures immediately beneath the superior vena cava. In patients with a good deal of collateral circulation to the lungs, this area is usually quite vascular and a number of vessels must be ligated. The innominate artery can usually be palpated just to the left of the trachea. After some of the tissues surrounding it are freed, a piece of umbilical tape is passed around the innominate artery by the use of a curved clamp. Traction on this tape and further sharp and blunt dissection will reveal the point at which the innominate divides into the common carotid and the subclavian arteries. If the innominate artery is to be used for the anastomosis, the first portions of the carotid and subclavian arteries are ligated, the proximal part of the innominate is occluded with a bulldog arterial clamp, and the innominate is cut across just proximal to the ligatures on its branches.

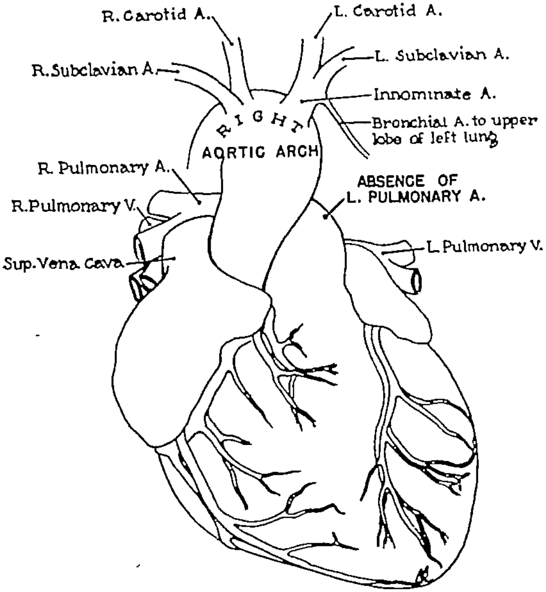

Fig. 1.—Showing the absence of pulmonary artery to the left lung. A branch of the innominate artery passed to the hilus of this lung.

It is highly desirable to use the subclavian artery rather than the innominate if its size is such that it will supply an adequate flow of blood to the lungs. It is now being used in the great majority of cases. The pleura overlying the subclavian artery is incised and a greater length of the vessel is exposed. The anatomical feature which makes it most difficult to expose the subclavian artery is the position of the vagus with its recurrent branch as it passes over the vessel. In some instances the vagus is pulled medially and the subclavian is exposed lateral to it. Usually, however, this maneuver is not necessary. The subclavian artery is ligated at the point where it gives off its first big branch, its proximal part is occluded with a bulldog arterial clamp, and the vessel is cut across proximal to the ligature. The section of the vessel is usually made in an oblique direction in order to expose the circumference of the end.

In an occasional patient the subclavian artery is so short that an anastomosis is difficult or impossible. This difficulty may sometimes be overcome by occluding both the innominate and carotid arteries temporarily with arterial clamps and removing the clamp from the subclavian artery. This step leaves the entire length of this latter vessel free for anastomotic purposes. However, even this maneuver may not give an adequate length of vessel. Under such circumstances it may be necessary to ligate and divide the subclavian artery, to ligate and divide the common carotid artery as high as possible, and to use the proximal end of the carotid for the anastomosis. It should be emphasized, however, that the carotid should be used only when the employment of the subclavian is impossible for it exposes the patient to the danger of a cerebral complication.

Regardless of which systemic vessel is chosen, the rubbershod arterial clamp with which it is occluded is tied in place with a heavy ligature around the two arms. This accomplishes two purposes: (1) It prevents the clamp from becoming dislodged, and (2) it affords a means by which one may make traction and thus approximate the systemic artery to the pulmonary artery. If, after having divided the systemic artery which is to be used, it appears that the arterial clamp is too near the free end, one may usually place another arterial clamp proximal to it and then remove the distal clamp.

As stated previously, the innominate artery arises on the left and is directed to the left when the aorta descends on the right. After the mediastinal pleura has been divided the innominate artery can usually be found without difficulty even though the superior vena cava is not present on the left to serve as a landmark. As a matter of fact, the most easily performed anastomosis is usually that in which the aorta descends on the right and the incision is made on the left. This is due to the fact that a greater length of the pulmonary artery is more easily accessible on the left than on the right. Furthermore, early branching of the left pulmonary artery is less likely to be encountered.

In every patient who has been explored there has been a branch of the arch of the aorta which could be used for the anastomosis. Such branches have not always been of the ideal size or length, but at least one or another could be used. This statement is not meant to imply that there have not been variations. For example, as far as could be discerned there was no innominate artery in twelve cases. It appeared that the four large vessels arose separately from the arch of the aorta. It was my impression that both common carotid arteries as well as one of the subclavians arose from the innominate very close to the arch of the aorta. At any rate, the fact that a second carotid arose from the innominate did not interfere with the use of the latter vessel or the subclavian for the anastomosis. In five patients the subclavian artery on the side of the operation arose directly from the aorta and passed posterior to the esophagus. This abnormal position did not interfere with the use of this artery for the anastomosis. This type of anatomical variation is shown in Figs. 2 and 3.

At times difficulties were encountered which were due to the short length of the systemic artery which it seemed desirable to use. A very short innominate artery was present in twelve cases. In fourteen other cases the subclavian branch of the innominate artery was quite short. It is significant, however, that there was in all instances a systemic artery which could be used for the anastomosis. In some cases the arteries were shorter or smaller than was desirable but nevertheless some systemic vessel could be used. The situation in this respect has been more satisfactory than that in connection with the variations in length and size of the pulmonary arteries. As stated previously, the carotid artery may be used if the innominate and the subclavian arteries are too short, but this is very undesirable and is rarely necessary.

In our earlier patients the innominate artery was used in a number of cases in which the subclavian artery would probably have been a better choice. Most children over the age of 2 have a good sized subclavian artery and one that is capable of transmitting a large quantity of blood. The subclavian should be occluded temporarily near its point of division in order to obtain a good idea as to the size of the vessel. The subclavian artery is being chosen more frequently and the innominate less often.

A few words may be in order regarding abnormalities of the superior vena cava. In two patients the superior vena cava was located on the left side but it entered the right side of the heart. In another, the superior vena cava was not visualized in the right side of the chest and it is assumed that it was on the left. In two patients a right superior branch of a pulmonary vein was connected to the superior vena cava.

The Anastomosis.—There are a number of reasons why an anastomosis of the end of a systemic artery to the side of one of the pulmonary arteries is to be preferred to an end-to-end suture, despite the fact that the former is more difficult. Among these are: (1) An end-to-side anastomosis allows the shunted blood to pass to both lungs; (2) the size of the opening into the side of the pulmonary artery may be made to correspond very closely in size to that of the end of the systemic artery, whereas there may be much discrepancy in an end-to-end anastomosis; (3) if following an end-to-end anastomosis in a patient who has atresia or marked stenosis a collapse of the lung develops on the side of the operation, the limited flow of blood through the opposite lung may not be sufficient to maintain life; and (4) if an end-to-side anastomosis is performed and if later in life the pulmonary blood flow appears to be inadequate, the operative procedure may be repeated on the opposite side, whereas it may not be possible to do this if an end-to-end anastomosis has been performed. If one of the pulmonary arteries is divided and an end-to-end anastomosis is made, it is doubtful whether the patient would tolerate occlusion

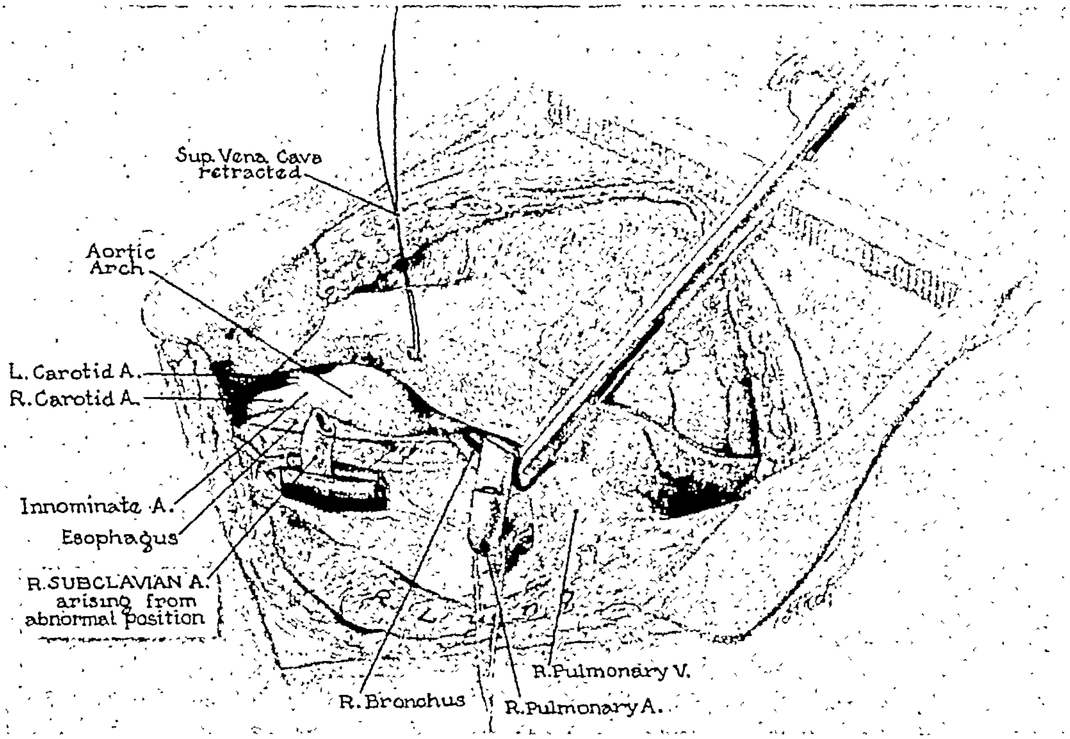

Fig. 2.—Showing a subclavian artery on the right which arose from the aorta rather than from the innominate and which passed posterior to the esophagus. Both carotids arose from the innominate.

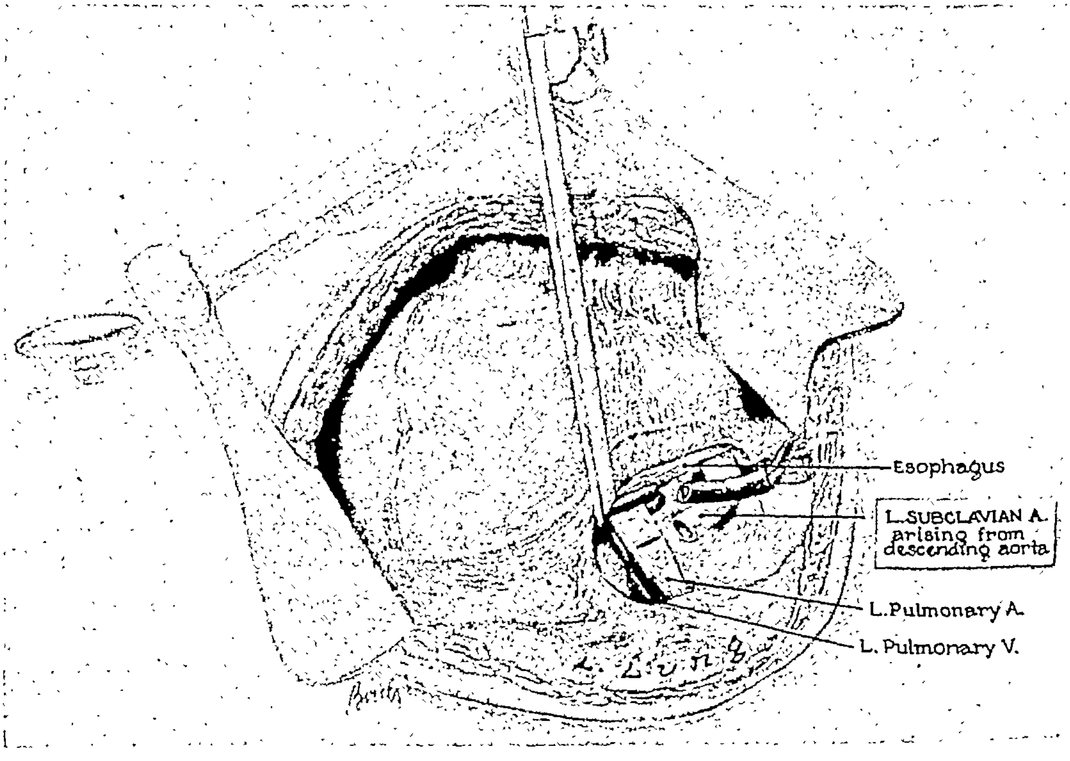

Fig. 3.—Showing the left subclavian artery in a patient with a right aortic arch. This vessel arose directly from the aorta instead of from the innominate and passed posterior to the esophagus.

of the opposite pulmonary artery for the length of time that is necessary for the accomplishment of an anastomosis. Despite these objections to an end-to-end anastomosis, it must be said that very dramatic results have followed the use of this method in most cases in which it has been used. Points in its favor are: (1) it may be performed more easily and quickly; (2) it may be used when there is a very short pulmonary artery and an end-to-side anastomosis is difficult or impossible; and (3) traction on the mediastinal contents is lessened. All things considered, however, the end-to-side anastomosis is preferred and has been used in most of the operations.

After the pulmonary artery has been dissected free of the surrounding tissues and after the systemic artery has been made ready, final preparatinos for the anastomosis are made. My remarks will be limited at this point to the end-to-side anastomosis. The right or left pulmonary artery is occluded proximally with a mechanical device, the tips of which are covered with rubber. This instrument has a long handle which makes it useful in stabilizing the mediastinum and in approximating the pulmonary artery to the systemic vessel. A photograph of two of the instruments is shown in Fig. 4.* Distal occlusion of the pulmonary artery is produced by traction on the pieces of braided silk which have been placed around the branches of the pulmonary artery. If the artery is unusually long, bulldog arterial clamps are placed on the branches. After proximal and distal occlusion has been produced, a transverse opening is usually made in the pulmonary artery between the points of constriction. This opening is made slightly bigger than the circumference of the end of the systemic vessel. Some of the adventitia surrounding the opening in the pulmonary artery is excised, but the pulmonary artery should not be weakened too much by removal of the adventitia around the entire circumference of the vessel. If the pulmonary artery is very much smaller than usual and if an end-to-side anastomosis is to be attempted, the opening in the artery should be in a longitudinal rather than a transverse direction.

After the incision in the pulmonary artery has been made, the anastomosis is begun. The suture material which has been employed in most of the operations is 00000 silk on small round needles (Deknatel). The suture is essentially a continuous one in which intima is approximated to intima. The suture includes the entire thickness of the wall of the artery. An attempt is made to place the sutures 1 to 2 mm. apart and approximately two-thirds of the posterior row is placed before the suture is drawn taut. Each end of this running suture is then tied to interrupted sutures which are placed at appropriate points. The continuous suture is begun again and the posterior row is completed, at which point another interrupted suture is placed. The anterior row of sutures is easier to place. It is usually interrupted at one additional point. The suture around the entire circumference of the anastomosis is in reality a single row of a continuous out-in and -over suture which everts intima and which is interrupted in several places in order not to constrict the lumen of the opening. It is just the opposite of the in-out and-over suture which is frequently employed in intestinal anastomoses in inverting the intestinal mucous membrane.

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*Made in three sizes by the Murray-Baumgartner Company, 5 West Chase Street, Baltimore, Md.

After completion of the anatomosis, the traction on the braided silk around the branches of the pulmonary artery is released and the instrument which has been used in occluding the pulmonary artery proximally is removed. If a spurting stream of blood comes from some point along the suture line, an additional suture is placed. If there is simply a slow ooze of blood, slight pressure is made and it will usually stop without the use of further sutures. Several minutes after all bleeding has ceased, the arterial clamp is removed from the systemic vessel. Usually no further bleeding occurs. If there is bleeding, the arterial clamp is replaced and the suture line is inspected again.

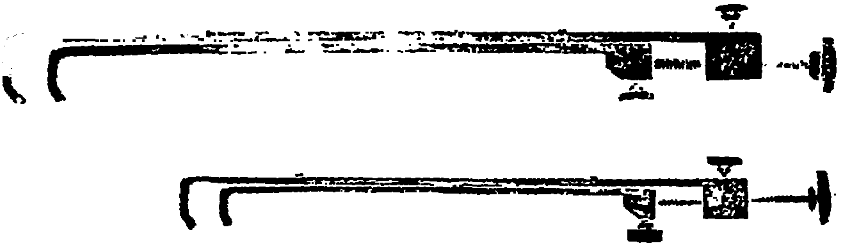

Fig. 4.—Showing two sizes of clamps which may be used for producing temporary occlusion of the pulmonary artery.

After the anastomosis has been completed and the constricting devices have been removed, a continuous thrill can usually be felt in the pulmonary artery. In many cases this can also be felt in the lung tissue at a considerable distance from the hilus. In infants and even in older children one may not feel a satisfactory thrill if the systemic arterial pressure is low.

After every effort has been made to be certain that the anastomosis is patent, including gentle massage of the opening, the mediastinal pleura is closed with interrupted sutures. The retractor is removed and the lung is partially inflated. Usually by this time the color of the patient has definitely improved. The third and fourth ribs are approximated with an encircling interrupted suture of braided silk or of some smaller material. The tip of a catheter is placed temporarily in the pleural cavity. The muscle, fascia, subcutaneous tissues, and skin are approximated with interrupted sutures of silk. Aspiration is carried out and the catheter is removed.

As indicated previously, it is necessary or at least advisable in an occasional patient to perform an end-to-end anastomosis between the proximal end of a systemic artery and the distal end of one of the two pulmonary arteries. This is somewhat easier than the end-to-side anastomosis. After choosing the parts of the two arteries which should be opposite each other, three interrupted sutures are placed at equidistant points. A continuous everting suture is then used in connecting these points.

Postoperative Treatment.—The patient is placed in an oxygen tent immediately after operation. The length of time that the administration of oxygen is continued varies from a few hours to a number of days. Penicillin therapy, which had been begun prior to operation, is continued for at least several days. The intravenous needle is left in place until the patient begins to take adequate fluids by mouth. The patient should be given adequate but not excessive fluids. If the arterial blood pressure declines, an immediate effort should be made to raise it by the giving of plasma or whole blood, because otherwise cerebral thrombosis or closure of the anastomosis might occur. If the patient presents evidence of weakness of the side of the body opposite the operation, heparin is given; otherwise, anticoagulants are not used. Dicumorol was given to the earlier patients in this series but its use has been discontinued. The patient is observed carefully for evidence of the accumulation of air or blood in the pleural cavity and aspiration is performed if indicated. If evidence of heart failure develops, a digitalis preparation is given.

In most patients the postoperative period is remarkably uneventful but in others it is very stormy. If death occurs, it is usually in the first forty-eight hours after operation. Some of the patients have been discharged from the hospital on the twelfth postoperative day, whereas others have remained in for considerably longer periods.

SUMMARY

The technique by which one may create an *artificial ductus arteriosus* in the treatment of pulmonic stenosis has been described. The ages of the patients have ranged from 2 months to 26 years. Operation has been performed on 144 patients with an over-all mortality rate of 22 per cent. This total includes several patients in whom the pulmonary artery was absent or was so abnormal that it could not be found at the time of operation.

An anastomosis was performed between the end of a systemic artery and the side of one of the two main pulmonary arteries in 118 patients with eighteen deaths, a mortality of 15 per cent. The subclavian artery was used in seventy-two of these with five deaths, a mortality of 7 per cent. The carotid artery was used in eleven cases with one death, a mortality of 9 per cent. The innominate artery was employed in thirty-six patients with twelve deaths, a mortality of 33 per cent. I have practically abandoned the use of the innominate artery in patients 2 years of age and older.

An end-to-end anastomosis was attempted in thirteen patients and there were six deaths. In five of the fatal cases the pulmonary artery was too small for a satisfactory anastomosis. In the remaining case the child was improved following operation but death occurred suddenly six months later from an unexplained cause.

AN ENDOBRONCHIAL BALLOON FOR THE CONTROL OF BRONCHIAL SECRETIONS DURING LOBECTOMY AND PNEUMONECTOMY

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DURHAM, N. C.

THE rapid progress in the development of surgical methods for the treatment of suppurative pulmonary diseases has made the need for adequate control of bronchial secretions during operation increasingly apparent. In a recent report of seventy-six cases of bronchiectasis treated by lobectomy or pneumonectomy, Bradshaw and O'Neill[1] record twelve deaths, six of which could be attributed to the transbronchial aspiration of pus expressed from the diseased lobe. Overholt and Wilson[2] report forty-five resections performed for pulmonary tuberculosis since 1942; contralateral spread occurred in 11.1 per cent. He states, "Contralateral spread is still the greatest hazard of pulmonary resection." Further reports serve only to substantiate the fact that it is a well-known and troublesome problem, especially for the anesthetist concerned with thoracic surgery. It is the purpose of this paper to present a new instrument for the control of bronchial secretions during the removal of infected lobes and to describe its successful use in experimental animals.

HISTORICAL

Most of the work in this field has been directed along three lines: namely, a catheter in the bronchus of the affected side for aspiration of secretions, a balloon in the affected side for obstruction of the secretions, or a combination of these two. Although aspiration by catheter has been in use for some time, possibly the first important step was made when Gale and Waters[3] in 1931 devised an endotracheal airway fitted at its distal end with a thin rubber cuff. The airway was inserted into the stem bronchus of the sound side and the cuff inflated so that it blocked both main bronchi at the carina. The most obvious criticism of this method was that the balloon could not be depended upon to block the affected lobe in lower lobe bronchiectasis; and, since there was no means of aspirating the pus, it could drain into the sound upper lobe. This method could be used satisfactorily, however, in pneumonectomy. In 1934, Archibald[4] reported seven cases in which he had successfully occluded the bronchus to the affected lobe by means of an inflatable balloon on a long coudé catheter. We have found that such a catheter cannot be depended upon to stay in place. Two years later Magill[5] devised a combined tracheoscope and anesthetic tube through which was inserted a suction catheter bearing an inflatable balloon. The anesthetic tube could be fitted with a cuff to hold it in place in the trachea while the balloon was placed in the stem bronchus. We have had no experience

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with this instrument, but it is our opinion that the balloon may be easily dislodged during operation. In 1941, Nosworthy[6] described the use of a balloon-endotracheal catheter to be used for pneumonectomy. This was inserted into the main bronchus on the sound side. Nosworthy noted the lack of a suitable instrument for use in lobectomies. He also mentioned Crafoord's[7] technique of bronchial tamponade for both pneumonectomy and lobectomy. This consisted in packing the stem bronchus with ribbon gauze kept in place by a wire rod; over this was threaded an endotracheal catheter. The most serious objection to these instruments and all others devised to date is their tendency to slip out of place at inopportune times; this obviously constitutes a serious hazard.

Many of the criticisms of these methods and of others not mentioned have been summarized in Beecher's[8] discussion of anesthesia in thoracic surgery. He recommends an endotracheal catheter made airtight at the face mask, the Trendelenburg position, and tracheobronchial suction by catheter. Blades and Graham[9] depend upon the upright position during operation to prevent secretions from entering the upper lobes. This method has been successful in seven cases, but they themselves raise the objection that in event of shock the head would have to be lowered; hence bronchoscopic aspiration would be necessary at once.

Thus it is apparent from the preceding discussion that there is still a need for some technique or instrument which will prevent the flooding of unaffected lobes with pus during operation.

CONSTRUCTION

After reviewing the methods tried in the past, it was decided that the most practical way to occlude the bronchus was by means of a balloon. We have therefore devised a double-lumen balloon-suction catheter (Fig. 1) for use in pneumonectomies and lower lobectomies. Early in our experimentation it was decided that it would be more practical to devise two instruments, one for obstruction of the lower lobes, and one for obstruction of the upper lobe, rather than to attempt to incorporate the necessary functions of both in one instrument. The instrument described in this report occludes the affected bronchus with a balloon retained in place by a special holding device, and provides aspiration distal to the balloon. The details of construction are as follows: The catheter is a double-lumen tube of the Miller-Abbott type, 4 mm. in diameter (12 French). At one end is mounted a small cylindrical balloon* approximately 6 mm. in diameter, 0.25 mm. in thickness, and 15 mm. in length. The balloon is inflated through one side of the double-lumen tube. The other lumen of the tube passes through the balloon and ends in a small perforated metal tip. Around the balloon is a small holding apparatus made of beryllium copper especially selected because of its flexibility, strength, and elasticity; this can be transferred within a few seconds from one balloon catheter to another. The over-all length of the assembled balloon, holding apparatus, and suction tip is 25 mm.; only the distal 10 mm., however, are in contact with the bronchial wall. As will be noted in Fig. 2, the distal end of each strip of the holding apparatus is doubly split, forming three projections, the middle one of which has a 1 mm. point. On inflating the balloon (Fig. 2, B) the points of the middle projections are pushed out into the mucosa, while the lateral projections are flattened out. When the balloon is deflated, all the projections spring back to their original positions, thus disengaging the points from the bronchial mucosa. On withdrawal of the deflated balloon, the two lateral projections protect the mucosa against the points of the middle projections. We first inflated the balloons with air. This was abandoned because of the danger of a sudden explosive rupture of the balloon. Water under mercury pressure was later adopted and found to be more satisfactory.

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*We are greatly indebted to the Miller Rubber Sundries Division of the B. F. Goodrich Co., Akron, Ohio, for supplying the balloons.

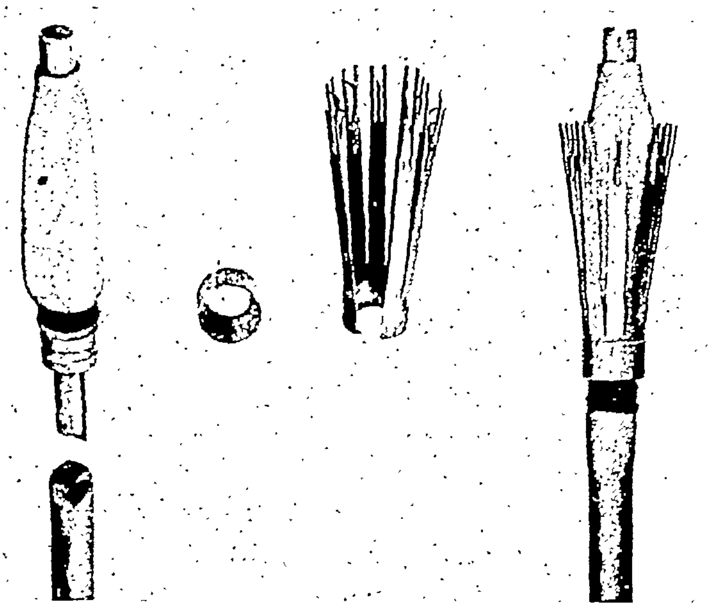

Fig. 1.—Authors' endobronchial balloon, consisting of a double-lumen tube with suction tip and balloon, surrounded by a holding apparatus.

In order to facilitate placement of the balloon in the bronchus, a brass carrier tube 50 cm. in length was devised, the distal 2 cm. of which was widened to fit snugly over the balloon and holding apparatus. This tube, which was threaded over the catheter, held the balloon encased in its tip. After the balloon was placed, a stylet of piano wire was used to dislodge it from the carrier tube.

The proximal end of the double lumen catheter is connected by a small, simple sleeve valve to the manometer and suction systems; the connection between the catheter and the two systems can be opened or closed by means of this valve (Fig. 3). It also affords a way of detaching the double lumen catheter from the pressure and suction systems and maintaining the pressure in the balloon; this facilitates quick removal of the bronchoscope following placement and inflation of the balloon. The entire apparatus including the sleeve valve will pass through a smooth bore 7 mm. bronchoscope.

PROCEDURE

Experimental operations were performed on a series of fifteen mongrel dogs, weighing from 22 to 54 lbs.

With minor variations, the following procedure was carried out in each dog. Anesthesia was produced by pentobarbital given intravenously in dosages of 30 mg. per kilogram. The tongue, pharynx, and larynx were anesthetized locally with pontocaine, and the site was selected for the balloon by bronchoscopy. The carrier tube holding the balloon and catheter was then inserted through the bronchoscope, projecting from its distal end a distance equal to the length of the balloon. Following this, the stylet was run alongside the catheter until it impinged on the base of the holding apparatus; with the balloon thus steadied, the carrier tube was withdrawn into the bronchoscope, leaving the balloon in place in the bronchus. The balloon was then inflated to the proper size as judged by the amount of water injected and the pressure in the manometer. With the manometer tube clamped, the sleeve valve was closed and the double lumen catheter was disconnected from the pressure and suction systems.

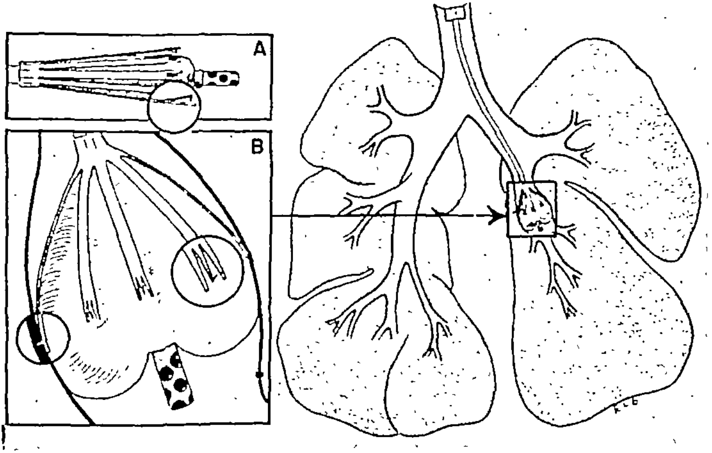

Fig. 2.—Diagram of balloon in lung specimen of a dog: (*a*) position of middle projection and point of each strip of holding apparatus when balloon is deflated; (*b*) inflated balloon showing the three projections of each strip, and lateral view of point embedded in the wall.

The bronchoscope, carrier tube, and stylet were then removed; an endotracheal tube, fitted with an inflatable cuff and attached to a metal T-tube, was slipped over the catheter, which was then reconnected to the manometer and suction systems. Suction was then applied, and atelectasis of the obstructed lobe or lobes was assumed complete when air ceased flowing through the suction bottle (Fig. 4). With the dog under positive pressure respiration, the lobe or lobes were removed using the individual ligation technique for the hilar vessels. After isolation of the bronchus was complete, the balloon was deflated and quickly pulled out through the endotracheal airway. The operator clamped the bronchus as he felt the balloon deflate and slip out. The wound was then closed and after voluntary breathing had resumed, the dog was turned over, and the procedure repeated on the opposite side.

A total of twenty-seven operations was performed on fifteen dogs including twenty-two lobectomies and five pneumonectomies, with no operative deaths.

EXPERIMENTAL RESULTS

At the beginning of this study it was apparent that the instrument must meet certain qualifications. It must: (1) have no harmful effects; (2) completely occlude the bronchus; (3) remain securely in place during operative manipulations; (4) have a wide margin of safety in its construction and operation, allowing complete control at all stages; (5) interfere as little as possible with anesthesia.

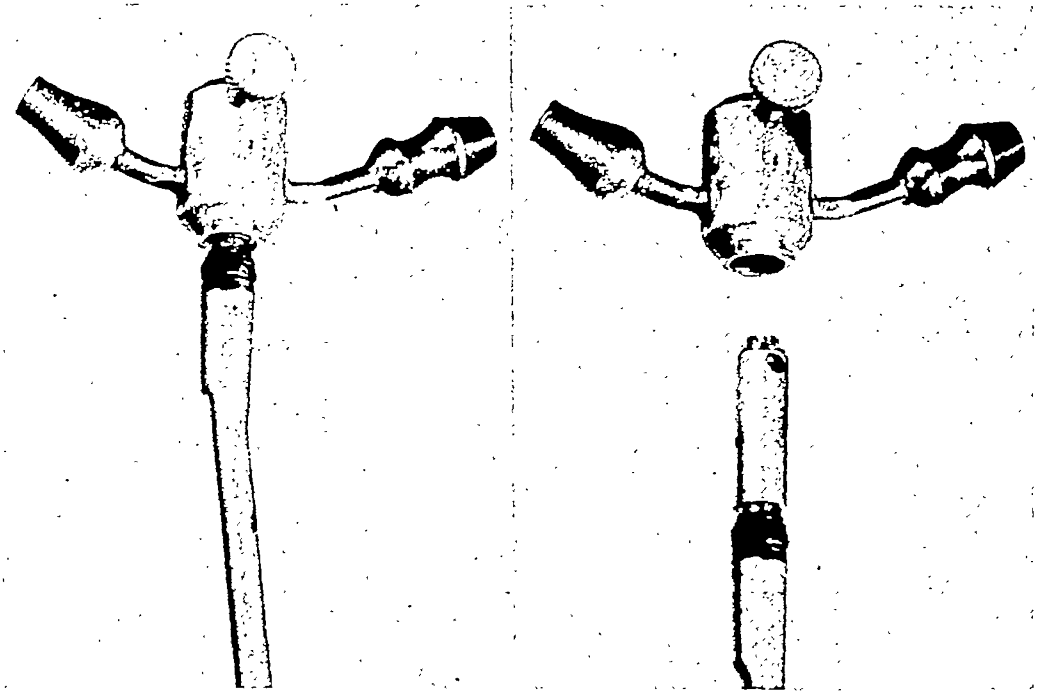

Fig. 3.—Sleeve valve and casing connecting balloon to pressure and suction systems. The valve is opened and closed through a 90° turn by means of the lock-screw in the outer casing; with the screw unlocked, the valve may be disengaged while still remaining closed and thus maintaining pressure in the balloon.

First, to test the effects of the balloon in the bronchus, balloons were inflated in the bronchi of two adult dogs anesthetized with pentobarbital and were allowed to remain in place for seven hours with the suction side of the tubing obstructed. Except for a slight bronchial irritation and consequent coughing on placement of the balloon, there were no effects on the respiratory or circulatory systems, nor on the general condition of the dogs. Following removal of the balloons, immediate bronchoscopy and subsequent direct examination of the autopsy specimens showed no bleeding points.

In order to test the completeness of occlusion, dyes were injected through the suction side of the tube into the bronchus distal to the balloon. Several mixtures were used, including Sudan III in mineral oil, India ink in mineral oil, and brilliant green. All specimens were removed *in toto* following the operations and the bronchi split open; in none of the specimens was dye present in any of the bronchi except the obstructed one. That the occlusion was complete was also indicated in another way: within a few minutes after applying suction to the obstructed lobe, air ceased bubbling through the water trap, showing that none was leaking around the balloon.

During each operation, tests were made to determine the holding power of the balloon in the bronchus. Extensive manipulation of the lobes failed to dislodge the balloon. Nothing short of a forceful pull on the double lumen catheter succeeded in dislodging it.

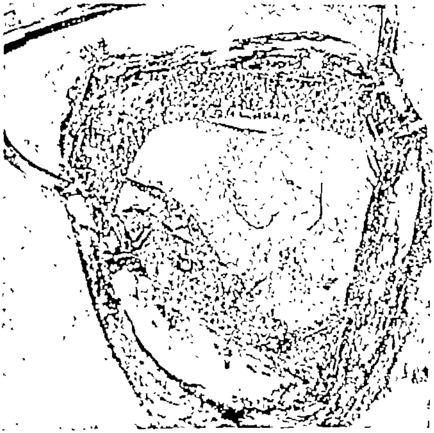

Fig. 4.—Photograph taken during an operation with balloon in left lower lobe bronchus; note atelectatic lower lobe and inflated upper lobe.

The limits of safety of the balloon were carefully studied. Attempts to break the balloon by overinflation while in place in the bronchus were unsuccessful; with the pressure increased to well over 400 mm. of mercury, the only noticeable effect was a slight dilatation of the bronchus and a bulging of the balloon into the lobe bronchi. None of the balloons were broken in this manner, although they had been used repeatedly. Deliberate efforts to break the balloon by trauma in the operative field failed.

The balloon was under complete control at all times, and its state of inflation and the completeness of occlusion could be quickly checked by noting the manometric pressure. It was found that the balloons required a pressure of 90 to 100 mm. of mercury for inflation; when in place in the bronchus, sufficient water (10 to 15 c.c.) was injected to raise the pressure to 200 mm. of mercury. Thus a pressure of 100 mm. of mercury was exerted on the wall of the bronchus. The pressure in the balloon is more than sufficient to hold the points of the holding apparatus in the bronchial wall, and since the resistance of the strips of this apparatus to bending is very slight there is very little hindrance to inflation of the balloon.

The small size of the catheter minimizes the interference with anesthesia as far as bulkiness is concerned; there is sufficient space left in the ordinary endotracheal tube to insert a small suction catheter should there be any secretions in the lower trachea. Local bronchial anesthesia may be used to lessen irritation and consequent cough stimuli. The fact that the pus is localized outside of the anesthetic field more than compensates for other minor interferences.

SUMMARY

A double-lumen balloon suction catheter for the control of bronchial secretions during lobectomy and pneumonectomy has been described. Its successful use in twenty-two lobectomies and five pneumonectomies in dogs demonstrates that (1) no harmful effects are produced, (2) complete occlusion of the bronchus is obtained, (3) dislodgement during operation is very unlikely, (4) a wide margin of safety is present, and that (5) there is little interference with anesthesia.

Clinical trials are now under way and will be reported later. It should be understood that this instrument has been designed for use only in pneumonectomies or lower lobectomies and not for use in upper lobectomies. Experimental studies are now being carried out on a similar instrument constructed primarily for use in upper lobe resections.

The authors wish to express their great appreciation to Dr. Deryl Hart, Professor of Surgery, Duke University School of Medicine, for his constructive criticism and interest in this problem.

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THORACOGASTRIC FISTULA AND MULTILOCULAR EMPYEMA COMPLICATING POST-TRAUMATIC DIAPHRAGMATIC HERNIA

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IN A paper presented at the 1946 meeting of The American Association for Thoracic Surgery, Lindskog and Lawrence[2] described two patients with diaphragmatic hernia following crushing injury to the thorax. Both patients had come under their care following the introduction of intercostal tubes into the herniated stomachs. Diagnoses of left sided empyemas had been made erroneously. In one case an empyema had developed following the drainage. Both patients were successfully treated with necessarily complicated operative procedures. In a careful review of the literature the authors were unable to find similar instances of diaphragmatic hernia and thoracogastric fistula following blunt injury of the thorax and surgical mistreatment. Clopton[1] had reported the case of a child with an eventration of the left hemidiaphragm. The patient had had a rib resection and tube drainage of the stomach through the greatly elevated diaphragm prior to coming under the care of Clopton. In this case thoracic wall and extrapleural abscesses were present. This patient also recovered.

During the recent war, penetrating and perforating thoracoabdominal wounds were frequently complicated by gastrointestinal injuries. At the time of primary operative treatment, the small intestinal and gastric wounds were customarily closed and wounds of the colon were exteriorized. Shaw,[3] who was working in a thoracic center, treated patients with thoracogastric fistulas. In these instances the repaired diaphragmatic and gastric wounds had broken down, and the hernias had followed. The patients were admitted to the thoracic center for further care.

Among the 511 patients admitted to a thoracic center* in England during 1944-1945 was one with a condition very similar to those reported by Lindskog and Lawrence.[2] In this instance it is possible that a gastric fistula was caused by a needle aspirating an hemothorax following a crushing injury of the thorax.

A 27-year-old sergeant was crushed against a brick building Nov. 15, 1944, by a large army truck, which had started in reverse by mistake. He suffered severe contusions of the thorax and was admitted to a hospital shortly after the injury. Red blood cells were present in the urine, but no further evidence of renal injury became apparent. A diagnosis of tension pneumothorax was made. After the removal of 550 c.c. of air from the left pleural cavity, the intrapleural pressures were reduced from plus 10 and plus 4 cm. of water to plus 4 and zero. Sanguineous fluid was aspirated from the left pleural cavity November 17, 18,

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*140th General Hospital.

19, and 22; 180 to 480 c.c. were obtained. Air was removed with the fluid November 20, and again December 5.

The patient was admitted to the center December 6. He was dyspneic and presented the general signs of infection. Roentgenographic examination (Fig. 1) showed the heart displaced to the right by the intrathoracic stomach and pneumothorax. The left tenth and eleventh ribs were fractured. Barium was given by mouth December 7. To the great surprise of the observers, the barium meal flowed rapidly from the uppermost portion of the stomach into the pleural cavity (Figs. 2 and 3).

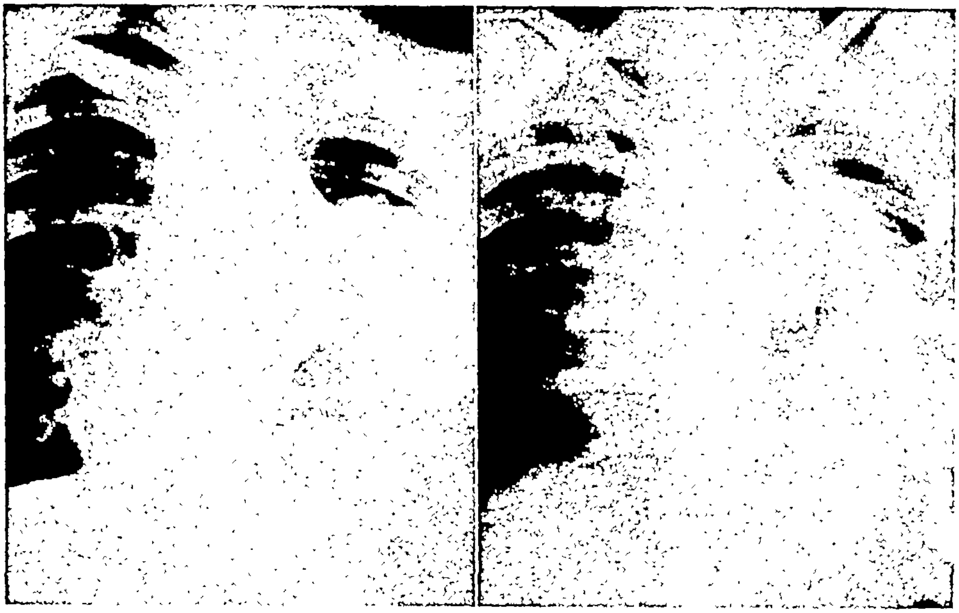

Fig. 1. Fig. 2.

Fig. 1.—Posteroanterior roentgenogram showing the heart displaced to the right by the intrathoracic stomach and pneumothorax.

Fig. 2.—Anteroposterior roentgenogram following ingestion of barium meal, which is seen in the stomach and free in the pleural cavity.

On December 8, with endotracheal ether anesthesia (Major Lawrence J. Schuhmacher, Jr., Medical Corps) the left thoracic cavity was opened through the bed of the resected ninth rib. The spleen, which lay posteriorly, was incised when the pleural cavity was opened. Brisk hemorrhage occurred, which was controlled only after ligation of the splenic vessels and removal of the organ. A large pocket of thick white pus lying on the diaphragm had been covered by the spleen. A segment of the eighth rib was removed to permit better exposure of the intrathoracic contents. The stomach was adherent to the posterior parietal pleura (Fig. 3) and displaced the lower pulmonary lobe upward. The omentum lay anteriorly against the pericardium, from which it was freed by blunt dissection. After the stomach was mobilized, the perforation 2 cm. in diameter was closed with two rows of fine silk Lembert sutures. The omentum, stomach, and the left lobe of the liver were then replaced into the abdomen. The diaphragmatic tear, which extended from within 2 cm. of the thoracic wall to the esophageal hiatus, was closed with overlapping by two rows of interrupted silk sutures. The multilocular empyema filled with thin sanguineous pus and barium, which had covered the stomach, was then broken down. The upper portion of the lower pulmonary lobe and the upper lobe were covered with plastic exudate, which had to be removed before the lung would expand. The thoracic wall was closed with interrupted sutures. Drainage of the pleural cavity was provided with a large rubber tube and water seal. A segment of the eleventh rib had been removed for insertion of the drainage tube. During the operation whole blood transfusions were given.

The patient's immediate postoperative course was stormy. Drainage of the pleural cavity was inadequate since the lung expanding posteriorly blocked the drainage tube leaving a large anterior air pocket. Fortunately the lung became adherent in the midthorax laterally and gradual re-expansion occurred aided by voluntary breathing exercises. Penicillin was given for some days in doses of 40,000 units every four hours. Several blood transfusions were necessary. The roentgenogram April 10, 1945 (Fig. 4), showed apparent complete re-expansion of the lung. The shadow at the base represented, however, a basilar empyema. April 23, the eighth rib was resected and a small empyema was dependently drained. At this operation four heavy silk sutures were removed from the diaphragm. In addition to hemolytic *Staphylococcus aureus*, which had also been cultured at the time of the first operation, *Aerobacter aerogenes* was grown. The cavity closed satisfactorily and only a small track remained at the time of the transfer of the patient to the United States June 2. He was in good physical condition with excellent thoracic respiratory movements.

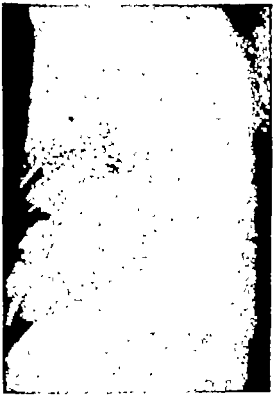

Fig. 3.

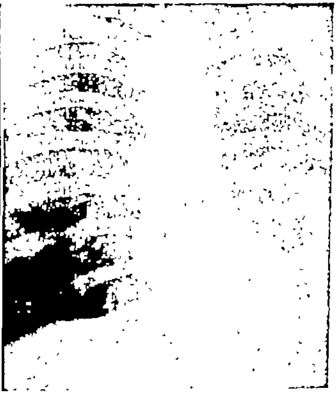

Fig. 4.

Fig. 3.—Left lateral roentgenogram after ingestion of barium meal; the stomach is seen lying posteriorly in the thorax with the barium passing superiorly and anteriorly.

Fig. 4.—Posteroanterior roentgenogram April 10, 1945, showing the re-expanded lung with evidence of basilar empyema pocket.

The case presents several lessons in the treatment of empyema thoracis. The abdominal organs, which had been intimately associated with the pus in the thorax, were restored to the abdomen without peritonitis developing; adequate penicillin therapy probably made this possible. More complete decortication and mobilization of the lung with the use of two or three intercostal drainage tubes would probably have facilitated complete and rapid expansion of the lung. The authors[4] found that more than one drainage tube was necessary after decortication. The advisability of using fine silk sutures for the repair of the diaphragm was well shown by the discovery of heavy silk sutures when the empyema was redrained.

SUMMARY

A case of thoracogastric fistula with multilocular empyema complicating a post-traumatic diaphragmatic hernia is reported. Diaphragmatic hernia must be considered following thoracic wounds and injuries and care must be used in aspirating or draining the pleural cavity following such injuries. In the reported case, aspiration may have caused the gastric perforation and the empyema.

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AMYLOID DISEASE OF THE LUNG TREATED BY PNEUMONECTOMY

Report of a Case

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Amyloid disease localized to the lower part of the respiratory tract appears to be an exceedingly rare condition and one which heretofore has not been recognized as a surgical disease of the thoracic organs.

GENERAL CONSIDERATIONS

Amyloidosis, or the presence of diffuse amyloid deposits in certain organs or tissues, has been widely studied and described in the past sixty years. Rokitansky is credited by Wichmann[1] with describing the microscopic and gross characteristics of the condition and with distinguishing for the first time amyloid deposits as a pathologic entity from other lesions of the organs concerned. As it is commonly used, "amyloidosis" refers to the multiple, abnormal, hyalinelike, intercellular deposits or collections, in the parenchyma of the liver, spleen, kidneys, adrenal bodies, and occasionally in other areas of the reticuloendothelial system, of an atypical protein substance which stains more or less specifically. In addition, these lesions are associated with or are secondary to a prolonged suppurative process and certain other primary disturbances, such as syphilis, Hodgkin's disease, and various types of leucemia. The lesions are most commonly seen among patients who have chronic pulmonary tuberculosis with cavitation or chronic suppurative osteomyelitis.

In recent years an increasing number of descriptive reports of unusual amyloid deposits have appeared. The deposits have been said to be either generalized or localized in other than the usual or characteristic locations, such as the spleen, liver, kidneys, or adrenal bodies. These observations have been referred to by many authors as indicative of "atypical amyloidosis."[2-4] Since the clinical picture was for the most part bizarre or simulated other more commonly recognized disturbances, the true nature of the pathologic process in most of the earlier cases was identified only by microscopic examination. In only a few cases was the true condition suspected before necropsy; the amyloid depositis were considered by some workers to be merely interesting incidental observations. In addition, there was a remarkable absence of a primary suppurative or debilitating disease which could be considered as the etiologic factor in the production of these deposits of amyloid substance.

Lubarsch[5] recognized this unusual or atypical form of amyloid disease and suggested the following criteria to distinguish it from the usual or typical forms: (1) an almost complete absence of amyloid substance in the organs

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most often involved in typical amyloidosis, such as the liver, spleen, and kidneys; (2) the presence of amyloid substance in organs and parts not usually involved by the process, such as the heart, skin, and lungs; (3) the occasional occurrence of tumorlike nodules of amyloid substance; (4) the frequent failure of the deposits to react to specific stains or tests for amyloid; and (5) the absence of a preceding or concomitant disease to which the presence of amyloid might be attributed. After review of many of the reported cases of the unusual forms of the condition, Reimann and associates,[6] in reporting another case, discussed the clinical, gross and microscopic features of the so-called atypical forms and suggested the following classification of amyloidosis: (1) primary (atypical or systematized); (2) secondary (typical); (3) tumor-forming (single or multiple, localized); and (4) amyloidosis occurring in association with multiple myelomas. These authors collected thirty-five cases of primary amyloidosis, in seventeen of which the distribution was more or less generalized. In the remainder, the process was localized largely to one or two organs. As a result of their study, Reimann and associates were able to observe certain rather distinct characteristics of each of the four aforementioned forms, although there was a tendency toward overlapping in certain cases. They pointed out, further, that whereas tumor-forming amyloidosis may be a type of primary amyloidosis, yet in several cases it was sufficiently localized from a clinical and pathologic viewpoint to warrant consideration aside from the generalized group. Subsequent writers[7, 8] have endorsed this classification. Several examples of all the four types previously mentioned herein have been added to the literature since the classification of Reimann and associates was made.[4, 9-11]

As indicated by the increased number of reported cases of localized amyloid "tumors," it would seem justifiable to consider as members of a separate group those patients in which the amyloid disease is apparently entirely localized to a certain organ or tissue or to a portion thereof. Additional examples of the latter were reported by New and Erich[12] and by Figi,[13] who recognized localized sessile, polypoid, or infiltrated masses of amyloid substance in the laryngeal or subglottic tissues unassociated with demonstrable deposits of amyloid substance in other parts of the body. Corbitt and associates[8] reported on a patient whose condition had been diagnosed preoperatively and who was successfully treated by segmental resection of tissue affected by localized amyloid disease involving a portion of the wall of the urinary bladder.

It is apparent that localized amyloid disease is a very unusual process but one which can be considered for surgical management when it is deemed suitable. In the known cases of this form of the disease, the respiratory tract and skin appear to be the commonest sites of involvement.

New,[14] in 1919, studied and discussed the clinical and pathologic features of amyloid lesions of the upper air passages. In 1938, New and Erich[12] reported the clinical aspects and method of treatment in eighteen cases of the localized amyloid tumors of the larynx. The clinical features of these lesions were sufficiently characteristic to warrant recognition; the presence of these masses altered laryngeal or glottic functions to such an extent as to warrant treatment. Surgical management was utilized to eradicate these nodular deposits.

On the other hand, we have encountered only three published reports[15-17] of localized amyloid disease of the lower part of the respiratory tract. In the case reported by Balser the condition was discovered at necropsy; there was marked tracheobronchial stenosis involving the lower portion of the trachea and major bronchi, associated with a secondary emphysematous, pneumonic, and bronchiectatic process which extended throughout both lungs. Symptoms had been present for nineteen years. There was a dense and rigid deposition of amyloid substance in the walls of the lower part of the trachea and of the bronchi and the lumens of both the trachea and bronchi were markedly narrowed. In Glockner's case, asymptomatic amyloid tumors involving the larynx, trachea, and large bronchi were discovered at necropsy, but no embarrassment of the lower part of the respiratory tract had occurred and no secondary processes were noted in the lungs. The only other pathologic observations of note in this case were a urethral stricture, benign prostatic hyperplasia, and cystitis. In Meyer's case, multiple nodules of amyloid substance of various size were scattered throughout both lungs, and appeared to be continuous with the bronchial wall in certain areas, but bronchial stenosis was not present. There were degenerative processes, but no deposits of amyloid substance were present in other organs. In addition, there was no evidence of a primary disease to which the amyloidosis could be attributed.

The following report of a case is presented as one of unusual interest. It is believed to be the first reported case in which localized amyloid disease involved the major bronchi and corresponding pulmonary vessels at the hilum of one lung, and the first in which pneumonectomy was successfully accomplished.

CASE REPORT

Clinical and Surgical Aspects.—A white man 35 years old, a welder by trade, registered at the Mayo Clinic on Jan. 10, 1946. He complained chiefly of cough of nine years' duration and of daily hemoptysis of a week's duration. He was admitted directly to a hospital for study. He said that in 1937 he had noted some pain of a pleuritic nature in the right side of the lower part of the thorax, without associated fever. In addition, morning cough, productive of purulent, occasionally blood-streaked sputum, had developed. Shortly after the onset of this illness he had been admitted to a hospital for observation and treatment. Bronchograms had been made, and the diagnosis of chronic bronchiectasis of the right lung had been made. Since that time symptoms had consisted of recurrent attacks of chronic, moderately productive cough, usually precipitated by "colds" and often associated with fever and chills and pleuritic pain. Sputum continued to contain occasional traces of blood. In 1940, more bronchograms had been made; the same condition had been found. The left lung had been found to be normal. In 1942, the patient had experienced a single, moderately severe episode of hemoptysis (approximately a cupful, or about 240 c.c.). No change in symptoms had been noted until a week before admission to the clinic, at which time acute coryza had developed, followed by a marked increase in the severity of the cough and in the amount of sputum raised. During the week prior to admission the sputum had been constantly bloody. The patient had become most apprehensive and insomniac from the fear of fatal hemoptysis. There had been no recent loss of weight or significant general weakness.

The patient's systemic history was noncontributory. He smoked about ten cigarettes a day. The general history was not significant. The patient's mother had died of tuberculosis at the age of thirty-five years. When we saw the patient on Jan. 10, 1946, he did not appear to be ill and did not exhibit evidence of a debilitating disease. The temperature was 99.6° F. (37.6° C.) and the pulse rate was 90 beats per minute. Results of examination imally on the main bronchus. The right pulmonary artery was included in the infiltrative process about the main bronchus. The thickness of the infiltration of the walls of the main bronchus reached 2.5 cm. in the region of the pulmonary artery. Several slightly enlarged lymph nodes were present in the hilus, but they did not appear to be abnormal. Tincture of iodine applied to the cut surface of the tumor did not give the characteristic gross color reaction for amyloid.

The remainder of the parenchyma of the lung peripheral to the mass exhibited the picture of a scattered pneumonic process throughout the middle and lower lobes of the lung. Evidence of diffuse bronchiectasis was not found, although a few scattered sacculated bronchioles containing thick, brownish, stringy exudate were noted in the middle and lower lobes.

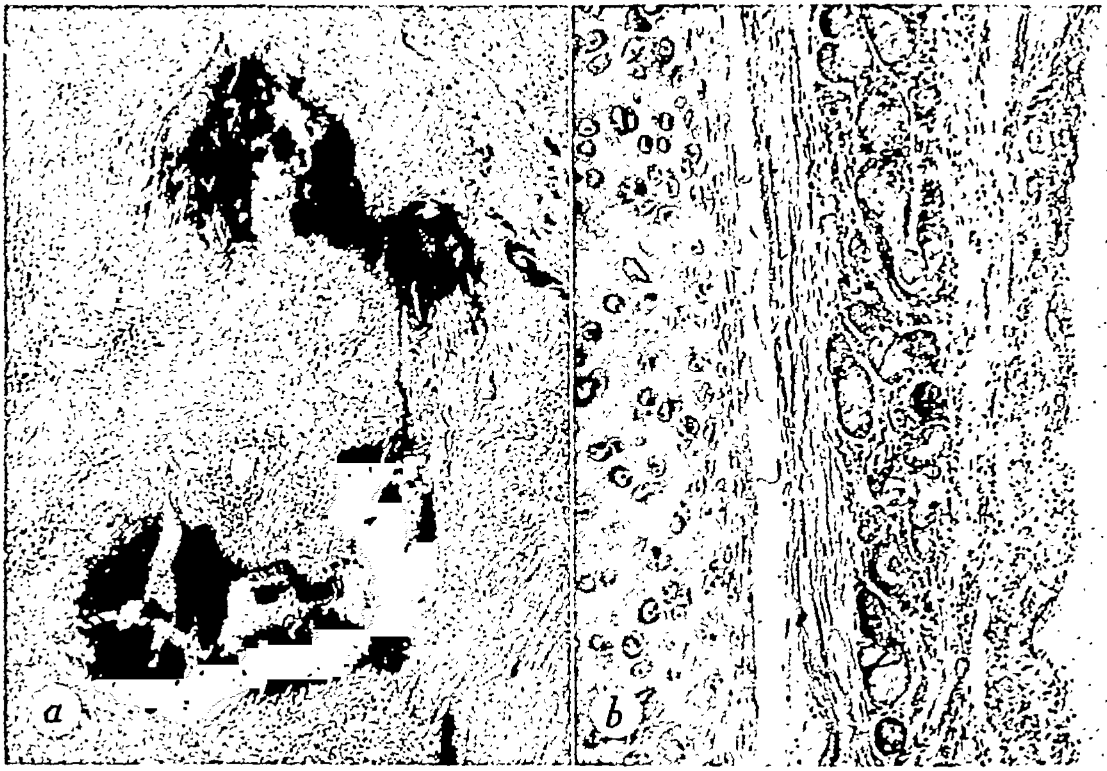

Fig. 4.—*a*, amyloid substance, with areas of calcification (hematoxylin and eosin ×60); *b*, bronchial epithelium and subepithelial structures in an involved portion of the bronchus; note the loss of ciliated stratified epithelium and replacement by single layer of cuboidal type of cells (hematoxylin and eosin ×100).

Histopathologic Aspects.—Sections through the walls of the bronchus and its branches extending into the tumor mass stained with hematoxylin and eosin showed the external coats of the main bronchus, its branches, and the surrounding connective tissue to be the seats of the pathologic process. The cartilaginous rings were intact. The dense, infiltrating mass which surrounded the bronchus and its branches and the pulmonary vessels at the hilus consisted of interlacing bundles of strongly eosinophilic homogeneous material which was very acellular and avascular. Scattered throughout were nests of inflammatory cells, consisting mainly of plasma and round cells, and lymphocytes. No giant cells were noted, although a few groups of fat-filled histiocytes were identified. Calcification of the homogeneous material was present throughout; it occurred both in small flecks and in masses of sizes up to 2 mm. (Fig. 4, *a*). The walls of the larger blood vessels were not abnormally thickened, but between the elastic fibers some of the homogeneous acellular material was seen. The changes in the mucosa of the main bronchus and its branches were limited largely

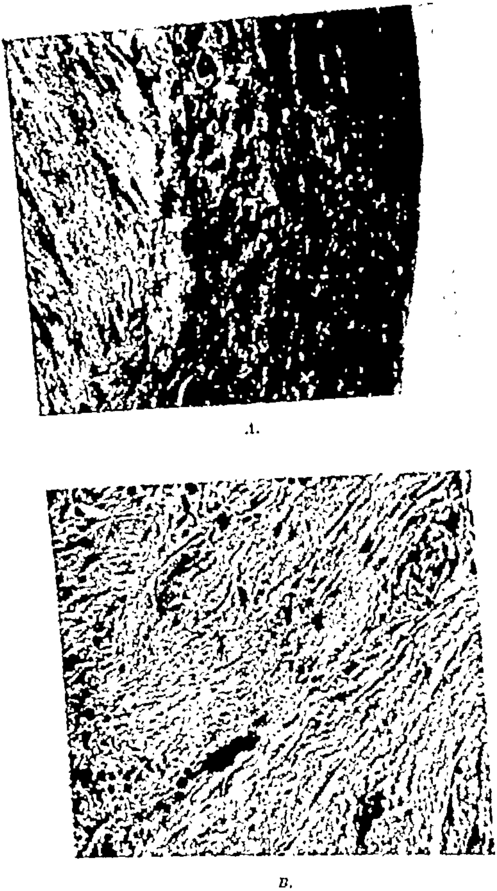

FIG. 5.—*a*, portion of the wall of a large branch of pulmonary artery and adjacent connective tissue involved by intercellular deposits of amyloid (methyl violet ×60); *b*, portion of the infiltration, showing lipoid degeneration of masses of amyloid (Sudan IV ×260).

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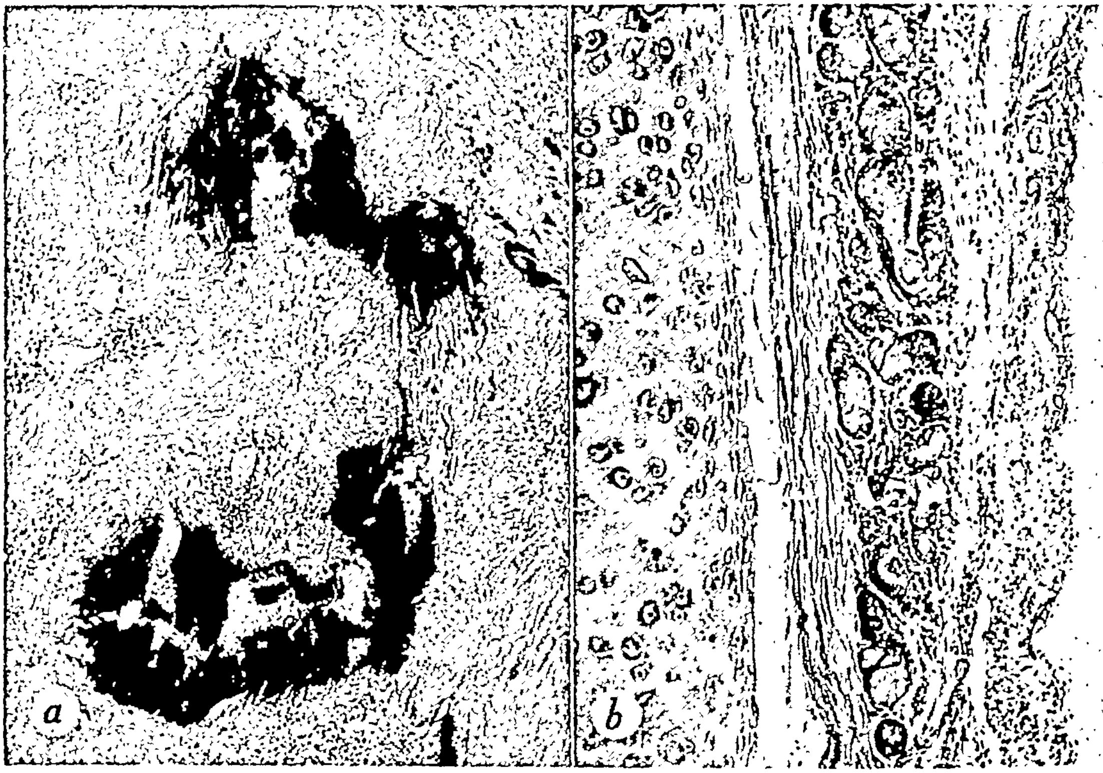

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Special staining of numerous sections through the involved bronchial walls, surrounding infiltration, and parenchyma of the lung was carried out with Congo red, methyl violet and Sudan IV stains. The hyalinlike intercellular material noted in the infiltrated regions previously described exhibited a lack of uniformity in staining reaction, whether it was stained with methyl violet or Congo red. When methyl violet was used, the color changes varied from gray to faint violet to brilliant violet; when Congo red was employed, the contrast in color between the amyloid substance and collagen was not uniformly distinct. Nevertheless, there were many portions throughout the sections which gave a diagnostic picture when each of these stains was used. The walls of the large arteries and veins exhibited irregular regions of amyloid infiltration, and in a few the entire wall stained characteristically for amyloid with methyl violet (Fig. 5, *a*) or Congo red. The smaller arterioles and veins in the mass were uniformly involved, as determined by the staining reaction. In fresh frozen sections many of these interlacing hyalinlike bundles appeared to contain numerous tiny, clear droplets. With the Sudan IV stain it was readily seen that these were fat-containing droplets within the bundles of intercellular amyloid material (Fig. 5, *b*). This phenomenon was seen in many places throughout the entire mass.

Sections of the parenchyma from beyond the gross limits of the tumor mass as stained with hematoxylin and eosin, revealed a scattered irregular, lobular distribution of a chronic exudative pneumonic process in the lower lobe and to a slightly less extent in the middle lobe. Near the hilar area, moderately chronic interstitial fibrosis and cellular infiltrations were seen. The alveoli of the lower lobe contained many macrophages filled with blood pigment.

The peripheral terminal bronchioles for the most part showed slight if any dilation, but mild inflammatory changes were present in the mucosa. A few of the medium-sized bronchioles exhibited mild-to-moderate dilatation, with associated intraluminal exudation and moderate peribronchial inflammatory infiltrations and fibrosis. The parenchymal blood vessels were essentially normal. Special staining of the parenchyma of the lung for amyloid substance revealed none, in either the alveolar or bronchiolar walls or blood vessels outside the gross region of tumefaction.

Lymph nodes situated at the hilus were uninvolved by the amyloid deposition, and showed only inflammatory changes with anthracosis.

COMMENT

Localized amyloid disease, particularly in the respiratory tract, can be responsible for sufficient alteration in the anatomy and physiology of the involved structures to be of clinical importance. In this case the clinical picture was essentially that of an intermittent, long-standing, low-grade, obstructing lesion of the major bronchus or its branches, at times associated with brief episodes of hemoptysis, without clinical evidence of the presence of a debilitating or suppurating disease. That the process did not produce complete or persistent bronchostenosis is evident by the absence of a significant degree of bronchiectasis or pulmonary suppuration distally. The history of the patient would indicate that some impairment of function and drainage of the middle and lower lobes of the right lung had been present for several years. It is interesting to note that relatively little anatomic narrowing of the bronchus and its branches could be demonstrated in the surgical specimen. The surgeon, however, noted at exploration that some degree of distortion of the hilus of the involved lung had been caused by the infiltrating mass, and it is likely that this could have produced some degree of kinking which in turn would have compromised the airway of a semirigid tube, such as a large bronchial branch. Furthermore, the loss of the cilia of the surface epithelium overlying the amyloid process in the main bronchus and its branches would suggest that a physiologic obstruction to the normal flow of bronchial secretion was present. There was no evidence of bronchial ulceration when the lung was examined in the laboratory. The source of the bleeding which resulted in the symptoms or produced the clot in the bronchial branch to the lower lobe, noted by the bronchoscopist, is best explained on the basis of extravasation from small or medium-sized bronchial blood vessels, the walls of which had been largely replaced and weakened by the described amyloid deposits.

The preoperative diagnosis in this case was that of the secondary associated disturbance. The repeated hemoptysis pointed strongly to a primary lesion involving the bronchial mucosa. Since the infiltration did not involve the mucosa or subepithelial portion of the main bronchus and its branches, it would have proved futile to attempt to determine further by bronchoscopic study or biopsy the nature of this extrabronchial lesion.

Certain other clinical considerations are immediately presented when a diagnosis of amyloidosis is made. In view of the reports in the literature of involvement of the mediastinum and lungs in cases of primary generalized amyloidosis, the question arises as to whether or not this process was only one of multiple similar processes in this patient. Additional clinical and laboratory studies made postoperatively, however, did not indicate that other deposits of amyloid substance were present. The experience of other authors would indicate that additional accessible cutaneous or submucosal nodules of amyloid substance should be evident at a relatively early stage in cases of the generalized atypical forms of amyloidosis.

It is interesting to observe that roentgenograms did not suggest the hilar infiltration in this case. This is especially remarkable in view of the fact that some degree of calcification was present in the mass. Yet, on subsequent review of the stereoscopic roentgenograms and the bronchograms, a roentgenologist said that he was unable to define the primary pathologic process in the roentgenograms.

Deposits of amyloid substance in tissues frequently are described in the literature as being grayish, translucent masses which yield a characteristic color reaction when iodine is applied to their cut surfaces. In this case, the gross picture did not show the classical picture, and did not suggest a typical form of such lesions. The extremely tough, leathery consistency of the mass as noted by both the surgeon and the pathologist was of much more significance in recognition of the nature of the process. The intercellular deposits of amyloid substance seen in the microscopic sections were, for the most part, arranged into bundles and, in some places, into irregular, small masses. The significance of the lipoid deposits within many of these amyloid deposits is not apparent, but may represent, in association with the calcification, a degenerative process. Increased fibrosis and cellular infiltrations throughout indicate that inflammation undoubtedly played a significant role in the development of the lesion in this case. Results of staining with Congo red and methyl violet, as specific stains for amyloid substance in tissues, were sufficiently positive to be confirming evidence of the nature of the intercellular material, but the variation in staining with both of these dyes was evident. This has been noted by other investigators and it has been thought that "preamyloid" or ground substances would not show the same staining characteristics as more mature amyloid. In our case it would appear that the presence of lipoid in many regions may have altered the staining properties.

In spite of several recent excellent experimental studies[18-21] on amyloidosis of the secondary type, the true nature of amyloid substance and the mechanism for its elaboration are still unknown. In recent studies, it is pointed out that the principal chemical structure of this material is that of a complex protein, probably conjugated with other proteins or polysaccharides.[22-23] Valuable additional information should be forthcoming when the chemistry of proteins is further investigated. The variation from the usual staining reaction seen in the so-called primary generalized form of amyloidosis and the tumor-forming localized form of the condition would suggest that perhaps some qualitative difference exists in the substance which is deposited in the presence of each of these entities as compared to the intercellular material present in the spleen, liver, kidneys, and adrenal bodies in the secondary form of amyloidosis. Whether the protein substance, called amyloid, is produced locally in the organs or tissues involved as a result of aberrant metabolic processes, or is deposited from the blood stream, is also undetermined. The process of chronic destruction and repair of tissue is apparent in all cases in which secondary amyloid disease is present. When experimental secondary types of amyloidosis are present, the fact that excessive stimulation with bacterial toxins and foreign proteins of the immunity mechanism of the organism plays an important role in the production of such deposits has been shown clearly by nearly all investigators. Generally speaking, therefore, amyloidosis is not a degenerative process, but is a local or generalized disturbance of protein metabolism.

No data are available to explain the factors involved in the production of amyloid deposits in tissues of mesodermal origin that are involved in either the primary generalized form of amyloidosis or in the localized amyloid "tumors." The origin of amyloid substance in the presence of these disturbances remains purely a matter of speculation regarding the role of a pre-existing inflammatory process. For example, the lower part of the respiratory tract frequently is the site of both acute or chronic inflammations, yet the apparent rarity of deposits of amyloid substance in recognizable amounts in these organs would militate against the fact that the localized form of amyloidosis is an end result of a chronic inflammatory process. Finally, further confusion is added to evaluation of etiologic factors of the production of amyloid when one considers the part played by myelomatosis in cases in which myeloma and amyloidosis coexist.

Of equal importance to the factors involved in the production of the amyloid disease is the consideration of the process of resorption of the abnormal depositis once they have occurred. That amyloid is resorbed from various organs has been observed both clinically[24-26] and experimentally[18, 19] in the secondary form of the disease. On the other hand, primary, generalized amyloidosis appears to be a progressively fatal disturbance from which no recovery has been reported. Localized amyloidosis seems to resemble more closely the primary or atypical form, so that in the presence of localized amyloidosis local resorption would not be expected to occur. Hence, surgical excision or fulguration must be the treatment of choice in the eradication of localized amyloidosis. The experience of laryngeal surgeons[18] at the Mayo Clinic has been that unless thorough excision of lesions involving the larynx and subglottic tissues is accomplished, results of treatment are indifferent and unsatisfactory.

The case we have presented is one of considerable interest in view of the increasing number of reports of localized amyloid disease for which surgical treatment has been utilized. It is hoped that this report will serve further to acquaint clinicians and surgeons with the several forms of amyloid disease, and to increase their awareness of the more atypical and localized forms. The latter, although apparently rare, can be the basis for obscure lesions in or about the lower respiratory part of the tract and, as in this case, may be amenable to some form of surgical excision. The localized form of amyloidosis also presents another entity of a sizable group of lesions for which exploratory thoracotomy is necessary if successful diagnosis, treatment, and determination of prognosis are to be achieved. At the time of surgical intervention, both the surgeon and the surgical pathologist must be sufficiently aware of the characteristics of localized amyloid disease to be able to assess properly such lesions for surgical treatment. The consistency of the tissue involved by these deposits is of diagnostic aid to the exploring surgeon, but the true nature of the process must be established by histopathologic examination. In recent months, at the Mayo Clinic, several patients who had amyloid tumors involving the larynx and urinary bladder have been encountered. In examination of such patients the endoscopist, in securing specimens of the lesions for biopsy, has been impressed by the toughness of the tissue and its resistance to cutting with ordinary forceps. With the aid of certain polychromatic stains, such as polychromatic methylene blue, the amyloid substance can be identified with relative ease in the fresh frozen microscopic section.

SUMMARY AND CONCLUSIONS

Amyloid disease, other than the commonly recognized typical form secondary to a chronic debilitating disturbance, is now being recognized as a clinical and pathologic entity. It may occur in a more or less generalized distribution or may be localized to a portion of an organ, particularly in the connective tissue elements and walls of the blood vessels of the involved structure.

Deposits of amyloid substance which form localized nodular tumors or infiltrations, although uncommon, may simulate other pathologic lesions and require surgical removal of either a portion of the organ or the entire organ.

The mechanism of elaboration of this abnormal intercellular material in localized masses is not known, and no evidence exists thus far that resorption of the deposits occurs either spontaneously or under the influence of conservative therapeutic measures.

The consistency of the involved tissues and the resistance of these tissues to cutting may be valuable diagnostic aids to the surgeon when he is dealing with unidentified masses. The staining characteristics of the amyloid substance are of considerable aid to the surgical pathologist in establishment of prompt positive identification.

A case of localized amyloid disease involving the main bronchus, its branches and the corresponding branches of the pulmonary vessels at the root of the right lung has been reported herein. Pneumonectomy was performed after thorough study in an attempt to establish the cause of the repeated episodes of hemoptysis.

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CASE OF MASSIVE CONGLOMERATE TUBERCULOSILICOSIS SIMULATING PULMONARY NEOPLASM

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THE case to be reported is that of a patient with a large intrapulmonary mass, which, although it had many of the characteristics of a neoplasm, was finally proved to be a massive conglomerate nodule of tuberculosilicosis.

In the literature we have found seven other microscopically proved cases of silicotic or tuberculosilicotic masses in which a clinical diagnosis of neoplasm had been made. There is also one not yet reported (Skavlem[10]). While others have been described, they all lack microscopic proof. The significant facts concerning these nine cases will be summarized.

CASE REPORT

The patient, P. G., a 58-year-old Polish male factory worker, consulted his surgeon, Dr. Gordon Holden, because of a poorly localized, lower right chest or upper abdominal pain of about two months' duration. There was no definite relationship of the pain to food, position, exercise, or deep breathing. The pain was accompanied by soreness across the upper abdomen as well as increasing weakness to the point of fainting, so that it was difficult for him to continue his work. Over a period of several months he had lost about 16 lbs. He gave no history of coughing, hemoptysis, or dyspnea. However, subsequent pulmonary function tests demonstrated a considerable decrease in his respiratory reserve.

Occupational History.—For a period of at least thirty years there was presumptive exposure to free silica dust. During the first ten years of this time he worked as a miner in a hard rock coal mine and for the next twenty years he was a molder in a foundry. For the last five years he worked chiefly as a factory elevator operator where there was probably no exposure to dust, but a portion of this time was spent in a sand pit used for testing machine guns.

Family History.—His father died at the age of 85 and his mother at 65, causes unknown. Three brothers, aged 70, 65, and 64, are living and well. There is no known history of cancer.

Physical Examination.—The patient was a fairly well-nourished white man of medium stature, who did not appear ill. No lymph nodes were palpable in the cervical, axillary, or inguinal regions. The chest was barrel-shaped and the right hemithorax appeared larger than the left. Expansion, although diminished, was equal on the two sides. Both hemithoraces were hyper-resonant. Over the right lower lobe posteriorly the breath sounds were more distant than elsewhere. There were no râles. Physical examination showed no other significant findings.

Laboratory Studies—

Chest roentgenograms by Dr. R. C. Hall (Figs. 1 and 2) gave the following findings:

"X-rays of the chest reveal a symmetrical thorax. The diaphragms are scalloped in appearance and are low in position. The cardiac silhouette appears normal. The lung fields are hyperventilated and there is an increase in the markings throughout the lung fields, as evidence of a fibrosis. There are also some small nodulations in the peripheral portions of the lungs. In the right lower lobe there is a dense tumor mass about 8 × 6 × 5 cm., which is in the posterior portion of the right lower lobe and, in the lateral view, overlies the spine. In the mediastinum there is a suggestive round shadow which may be a

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metastasis. Impression: tumor in right lower lobe; suggestion of metastasis to a mediastinal node; diffuse pulmonary fibrosis, which is suggestive of exposure to dust."

Bronchograms by Dr. Karl Gruppe, following bronchoscopy, showed distortion of the right lower-lobe bronchi posteriorly in the region of the tumor mass with some blocking.

Pulmonary function studies were done by Dr. George Wright, Nov. 29, 1943. Fluoroscopy showed that both diaphragms and ribs moved equally and normally. The mediastinum remained in the midline. There was no evidence of bronchial obstruction, either complete or partial, as determined by the presence of trapped air, and the tumor mass within the lung did not pulsate.

Resting minute ventilation was 10 L., and maximum breathing capacity was 84 L. Bronchospirometry results follow:

	RIGHT (%)	LEFT (%)
Minute ventilation	53	47
Oxygen absorbed	52	48
Carbon dioxide output	50	50

Blood: Red blood cells were 4,800,000; hemoglobin, 96 per cent; white blood cells, 6,200. The differential count was polymorphonuclear neutrophiles, 54 per cent; lymphocytes, 35 per cent; monocytes, 7.5 per cent; eosinophiles, 3.5 per cent; with nonfilaments, 20 per cent. Sedimentation rate was 14 mm. in one hour (Cutler method). Wassermann was negative.

Urine: The specific gravity was 1.017 to 1.025. Urine was consistently negative for sugar, albumin, casts, and red blood cells, with an occasional white blood cell.

Sputum: None was produced for examination.

Electrocardiogram was normal except for prolongation of the P-R interval.

A diagnostic bronchoscopy revealed no direct evidence of tumor. The only significant finding was some obstruction of the dorsal bronchus to the right lower lobe.

A previous bronchoscopy reported by Dr. Gruppe revealed "a suspicious thickening of the septum between the axillary segment and the basal bronchi of the right lower lobe."

Upon completion of the studies, it was concluded that the patient had a pulmonary neoplasm of undetermined type and the presence of silicosis was considered to be an incidental finding. The malignancy of the tumor was questioned because of its size, sharp outline, and apparent lack of lymph node involvement.

Pneumothorax on the right was instituted as preliminary to exploration. Four days later, on Dec. 4, 1943, the chest was opened. The lung was free except for a few adhesions, mainly to the diaphragm. The parenchyma was studded with many small hard nodules. The apex of the upper lobe was shrunken and suggested the presence of an old tuberculosis. A very firm, rounded mass, 6 or 8 cm. in diameter, was situated in the apex of the lower lobe. Because of obliteration of the major and minor fissures, it was impossible to determine the extent of invasion of the tumor. Mediastinal nodes were palpable, and although they were soft, the possibility of metastases could not be excluded. It was decided, therefore, that a pneumonectomy should be performed. This was carried out by the usual technique of individual ligation of the hilar structures and closure with interrupted silk sutures of the bronchial stump, which was then pleuralized.

Postoperatively, sterile fluid was removed from the right pleural space on a few occasions to neutralize the pressure. Except for the development of an attack of influenza on the fifteenth postoperative day, the patient's course was smooth. A sputum examination during this attack was negative for acid-fast bacilli both by direct examination and guinea-pig inoculation. He returned to his home on the thirty-fifth postoperative day.

Because of mediastinal displacement to the right and the presence of silicosis in the left lung, a two-stage thoracoplasty, right, was performed on March 23, 1944, and April 14, 1944. Since recovery from these operations he has regained his weight and strength. There continues to be some disability, chiefly because of dyspnea and also because of discomfort in the right chest.

Pulmonary function studies (Table I) were performed by Dr. Wright (1) before pneumonectomy; (2) postpneumonectomy but before thoracoplasty; (3) post-thoracoplasty.

The surgical specimen was examined by Leroy U. Gardner and William H. Carnes of the Saranac Laboratory (Figs. 3, 4, and 5) and showed the following:

Gross: There was black pigmentation, marked diffuse emphysema, and an occasional silicotic nodule from 1 to 5 mm. in diameter; there was a large, oval, 8 x 4 cm., sharply circumscribed, black, conglomerate, tuberculosilicotic mass in the hilum of the lower lobe; a discrete 5 mm. calcified nodule was in outer layer of conglomerate mass, and a completely calcified hilar lymph node.

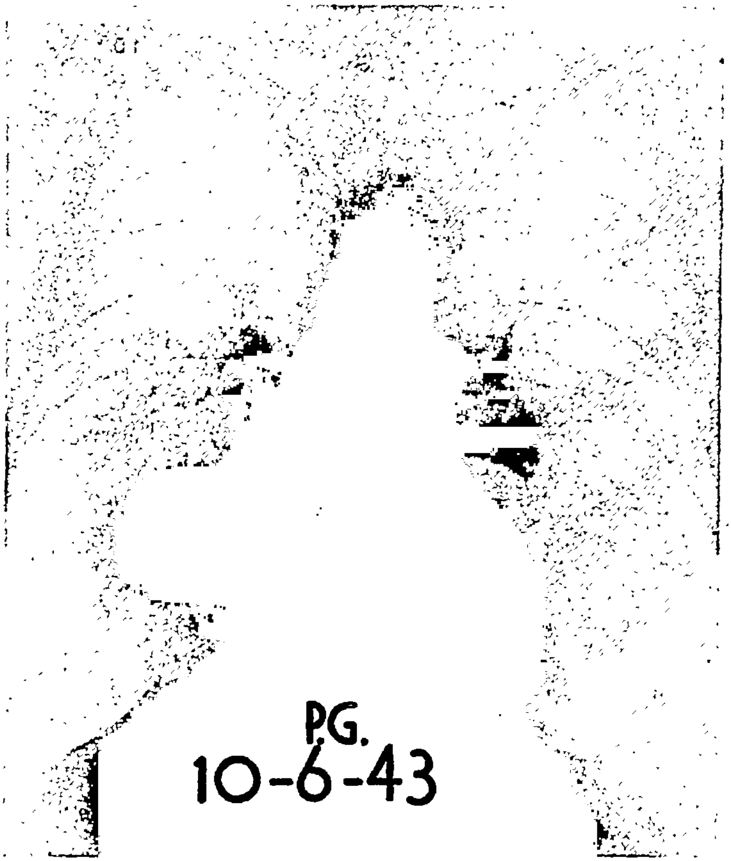

Fig. 1.—Posterior-anterior roentgenogram showing the tumor in the right lower lobe and an increase in markings throughout both lung fields.

Microscopic: There were scattered small hyalin silicotic nodules in all parts of the lung; the large mass at the hilum of the right lower lobe was a typical conglomerate lesion with some areas of granular caseous necrosis but no fresh cellular or epitheloid cell lesions; and a very occasional acid-fast bacillus was found in the caseous mass.

Diagnosis: Silicosis; conglomerate tuberculosilicotic lesion of right lower lobe; and healed calcified primary tuberculosis complex.

SUMMARY OF REPORTED CASES

In Tables II and III there is a compilation of the significant data on the nine reported cases of verified conglomerate silicotic nodules simulating neoplasm. Rendich and Camiel[9] recently reported two cases of their own and discussed five others. Of these seven we have accepted six as proved. The other case they mentioned, one reported by Piaggio Blanco and associates,[7] has been omitted here because microscopic proof was lacking. Another case has been reported by Zech.[13]

The case here reported is number eight In addition, Dr. John H. Skavlem[10] reported to us that late in 1943 he had a similar case in which lobectomy was performed for a tumor which on microscopic examination proved to be silicotic.

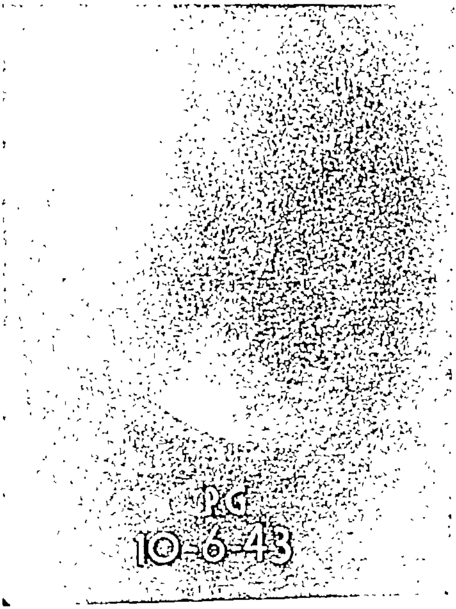

Fig. 2.—Right lateral roentgenogram showing tumor in lower lobe.

He has since written[11] that, due to personnel upsets occasioned by the war, he has been unable to date to make a more complete report. This would seem to be the ninth proved case. In addition to the reported proved cases, there may be others.[7, 13] Gardner[3] states that conglomerate lesions are common in silicosis, but that because of their bilateral or "bat-wing" appearance, together with their ill-defined outline and their appearance in lungs with widespread silicosis, the typical cases are seldom diagnostic problems.

A summary of the cases here listed reveals that the most common symptom is dyspnea, at first noted only on exertion, but gradually progressing. Loss of weight was noted in six patients. Expectoration, when noted, was scanty, and there was no record of hemoptysis.

The average age in the eight patients at the time of hospitalization was 52 years, with extremes of 34 and 73 years.

In the eight patients having pathologic examination, tuberculosis was proved in 50 per cent. In only one patient, however, were tubercle bacilli found clinically (Case 1), and then only by gastric analysis. Sputum, if present, was usually scanty. This was observed also in some of the other reported probable cases, such as those of Piaggio Blanco and associates[7] and Minet and associates.[6]

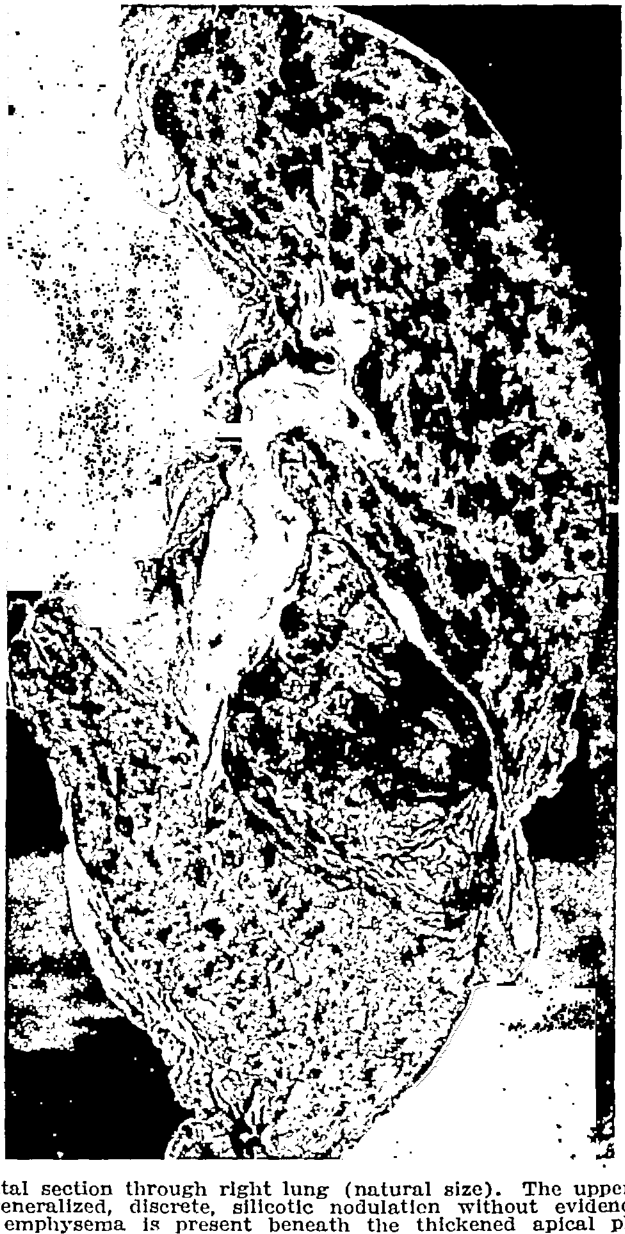

Fig. 3.—Sagittal section through right lung (natural size). The upper lobe presents the usual picture of generalized, discrete, silicotic nodulation without evidence of infection; a localized focus of emphysema is present beneath the thickened apical pleura. The middle lobe is not included in the plane of section. A large, ovoid mass can be seen occupying a portion of the lower lobe. This is bounded above by the fissure and below by its essential membrane. This "tumor" is black, hard, and rubberlike, except in places where it is undergoing caseous, softening. Mesially it includes an enlarged, calcareocaseous, silicotic lymph node which adjoins the bronchus and dislocates that structure. Elsewhere the lower lobe is free from evidence of infection. Mesially there are discrete black silicotic nodules and, in the costophrenic regions, extensive emphysema.

Bronchoscopy was performed in Cases 3, 4, 5, and 8, with negative findings. Aspiration biopsy was performed by Gut[4] in Case 5, and has been discussed by Piaggio Blanco and associates,[7] but it has not resulted in the making of a proper diagnosis in a single instance.

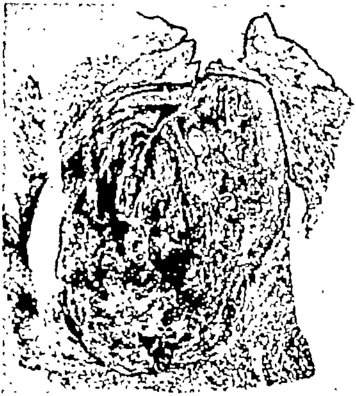

Fig. 4.

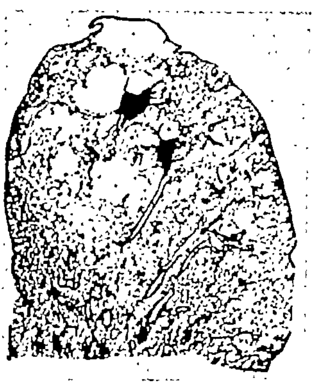

Fig. 5.

Fig. 4.—Histologic section of conglomerate lesion (magnification 1:1). This shows the *sharply defined borders with absence of inflammatory reaction in the surrounding lung.* The center of the mass is composed of diffuse and nodular hyaline fibrous tissue heavily pigmented with coal dust. Areas of necrosis are of lighter shade. No calcified foci are included in the plane of section.

Fig. 5.—Histologic section of the apex of the upper lobe (magnification 1:1). Emphysematous blebs can be seen surrounding two black, silicotic nodules. There are many small, discrete nodules widely scattered through the section. There is also considerable linear pigmentation in the pleura and the walls of blood vessels.

DISCUSSION

The significant fact which is demonstrated by the present case and the series of eight others briefly reviewed is the confusion which can arise in making a differential diagnosis between conglomerate tuberculosilicosis and pulmonary neoplasm. The problem of differential diagnosis is simplified when the masses are bilateral as is usually the case, and particularly when the masses occur on a background of generalized nodulation.[3] If one is fortunate enough to have serial roentgenograms over a period of years, confusion is not likely to occur. Furthermore, the behavior of the shadows in such a series is perhaps the most reliable index of activity of the process. In the presence of silicosis a conglomerate nodule is much more likely to occur than is a carcinoma.[2] On the other hand, Pohle and Ritchie[8] comment that in their case "roentgenological diagnosis of neoplasm was justifiable even in the light of post-mortem findings."

In two of the cases reviewed here (Cases 3 and 8) the masses were unilateral. This fact alone is confusing. In addition, when a case is first seen with a well-developed nodule and there have been no previous films, diagnosis may be difficult. Piaggio Blanco and his co-workers[7] report that "many cases of silicosis of the lung have been operated upon with the mistaken diagnosis

TABLE I. RESULTS OF STUDIES OF PULMONARY FUNCTION

PULMONARY FUNCTION STUDIES	PREOPERATIVE 11/27/43		POST-PNEUMONECTOMY 3/20/44		POST-THORACOPLASTY 10/13/44	
	LITERS	%	LITERS	%	LITERS	%
Maximum breathing capacity	84.		54.		55.	
Lung volume:						
Total	7.11	100	3.74	100	3.37	100
Vital capacity, available	4.98	70	2.21	59	1.88	56
Vital capacity, used	4.28	60	2.19	58.5	1.84	54.5
Mid capacity	3.54	50	2.40	64	2.09	62
Reserve air	1.41	20	.87	23	.60	18
Residual air	2.13	30	1.53	41	1.49	44.5

TABLE II. PROVED CASES OF SILICOTIC LESIONS SIMULATING NEOPLASM

CASE	AUTHOR	LOCATION	SYMPTOMS	CLINICAL DIAGNOSIS
1	Rendich and Camiel	Bilateral: right consolidation of base; left, smaller area in middle	Cough following broken ribs, loss of weight, weakness, occasional hemoptysis	Carcinoma of right lung with metastasis to left
2	Rendich and Camiel	Bilateral: right, upper; left, upper and smaller	Recurrent attacks of nocturnal dyspnea, loss of weight	Lymphoma or carcinoma of lung
3	Bradshaw and Chadoff	Unilateral: right, area size of half dollar at hilum; left, clear	Cough, loss of weight, and dyspnea for 9 mo.	Lung abscess—later, pulmonary carcinoma
4	Pohle and Ritchie	Bilateral: right, large, irregular mass into lower lung; left, smaller mass	Dyspnea on exertion and weight loss	Bronchogenic carcinoma
5	Holman and Pierson's Case #7	Bilateral: Right, round circumscribed density at base; left, large mass, base.	Numbness and weakness of legs for one year	Tumor or abscess with pneumonoconiosis
6	Gut	Bilateral: left, "fist-sized shadow" upper portion; right, smaller shadow, middle field	Dyspnea for 3 to 4 years, cough and expectoration, chest pain, loss of weight, sweats	Carcinoma of lung, either primary or metastatic
7	Zech	Bilateral: left, infraclavicular, sharply defined, oval shadow size of palm of hand; right, slightly smaller shadow present for 7 years	Increasing weakness and psychomotor instability	Bilateral lung tumor
8	Woodruff and Kelley	Unilateral: right, 8 x 4 cm. oval, sharply defined shadow, lower lobe; left, clear except for increased markings	Weakness, loss of weight, chest pain	Tumor right lower lobe, type undetermined
9	Skavlem	This patient had lobectomy for tumor which proved to be a massive silicotic lesion.		

of carcinoma and the correct diagnosis established only after the histologic study of the specimen removed.'' While this was true in the case reported here, as well as in that of Bradshaw and Chadoff[1] and also the one of Skavlem,[10] no others have been discovered in the literature. Zech[18] states that Conrozier and Magnier reported tumorlike shadows in thirty miners. In any case of silicosis with a coexisting discrete pulmonary shadow, a diagnosis of conglomerate tuberculosilicosis should be entertained.

The relationship between tuberculosis and silicosis is close. Gardner[3] feels that the large, conglomerate, silicotic lesions almost always show tuberculosis, and points out the frequency with which bacilli ultimately appear after these lesions attain maturity, even though the sputum has been negative for a long time. (In the present series sputum was recorded as positive in one instance and at post-mortem examination only 50 per cent were felt to be tuberculous.)

In some instances these lesions become caseous and excavate. Such an occurrence introduces the term of "phthisis atra," a name in the older literature "used to designate cavity formation in high-grade anthracotic lungs of miners."[12] The case of Holman and Pierson[5] (Case 5, Table II) illustrates this entity. The patient was a quartz miner with marked pneumonoconiosis and bilateral cavities and, therefore, a case of "phthisis atra."

In making a diagnosis of conglomerate tuberculosilicosis, the most important factor would seem to be a history of prolonged exposure to silica. Table III shows that the actual duration of exposure is known in five patients and averaged twenty-four years. In only one case could no exposure be traced, and that fact alone would cast doubt upon the diagnosis if it were not that chemical analysis of the lung tissue revealed a tremendous increase in silica content.

Although exposure is usually prolonged, symptoms may not develop until years after exposure has ceased. Rendich and Camiel's[9] first patient had a history of 25 years' exposure, but had not been exposed for twenty-four years before hospitalization.

TABLE III. DURATION OF EXPOSURE TO DUST

CASE	AGE	DURATION OF EXPOSURE AND TYPE OF DUST	YEARS SINCE EXPOSURE	TREATMENT AND PROGRESS OF DISEASE
1	73	25 years coal dust	24	Terminal admission, no treatment
2	46	Coal miner and steel worker unknown number of years	Unknown	Terminal admission, no treatment
3	34	12 years coal dust	None (?)	Two courses of x-ray therapy followed by extension of disease bilaterally; pneumonectomy, contralateral pneumothorax, death
4	36	No history of exposure to dust	Unknown	X-ray therapy; 2 months later showed bilateral increase; additional treatments did not prevent steady decline
5	58	28 years quartz miner	Unknown	Treatment limited to accompanying anemia; death in 6 months
6	56	Several years in pottery factory	20+	Dyspnea noted 4 years before admission; condition stationary for 8 months under observation; death 3 days following admission
7	61	25 years miner in Germany (Herner Region)	10	Eight years before admission and 2 years after cessation of exposure, applied for pension as invalid; diagnosis of pulmonary tuberculosis made on physical examination, never confirmed by x-ray; no expectoration. Admitted for terminal care; irrational; no specific treatment.
8	58	10 years hard rock coal miner; 20 years as moulder in foundry	5+	Pneumonectomy; later, thoracoplasty; dyspnea more evident following pneumonectomy.
9		Facts unknown[11]		

TREATMENT

We know of no specific medical treatment beneficially affecting silicosis. Although roentgen therapy was employed in Cases 3 and 4, it was based upon the mistaken diagnosis of pulmonary carcinoma. Its use was followed by extension of the lesion in each patient. Surgery is of limited value. Most patients with fairly extensive silicosis are quite dyspneic, so that any procedure involving a sacrifice of lung tissue will necessarily increase the disability. With the present method of measuring individual lung function, it is possible to predict quite accurately the expected disability following pulmonary resection. The removal of a conglomerate tuberculosilicotic lesion by lobectomy or pneumonectomy may at times be an impossible or at least a futile procedure. In those instances where the tumor formation is unilateral and the patient's respiratory reserve is adequate, as in our case, we believe exploration and resection is indicated for two reasons. First, diagnosis is not so certain that malignancy can be excluded. This was true in our case and in case 4.[8] Second, in certain instances these tumors undergo necrosis and cavitation. It would seem desirable to prevent this if possible.

SUMMARY

A single case of conglomerate tuberculosilicotic nodule is presented, another is mentioned, and the seven previously reported cases are discussed briefly.

Whenever a lung tumor appears in a person who has been exposed to large amounts of silica, the possibility of a conglomerate tuberculosilicotic nodule should be considered.

We are indebted to Dr. L. U. Gardner and the staff of the Saranac Laboratory for the preparation and study of the pathologic material presented in this paper.

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SPONTANEOUS RUPTURE OF THE ESOPHAGUS

Report of a Case with Recovery

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SPONTANEOUS rupture of the esophagus is of rare occurrence. It is generally accepted to be fatal when it does occur. For that reason it is worth while to record the following case in which the patient went on to complete recovery. By the term "spontaneous rupture" is meant the rupture of an esophagus presumed to be previously normal.

In 1943, Klein and Grossman[7] reviewed the literature on this subject and found forty cases reported with a mortality rate of 100 per cent. Since then, Collis, Humphreys, and Bond[3] have reported one and Eliason and Welky[4] three more. None of these patients recovered.

While a great many patients with esophageal perforation due to foreign bodies or instrumentation have recovered, to date no patient with spontaneous rupture has been reported that survived. Graham,[6] in a footnote in the *Yearbook of General Surgery* for 1944, states that he has seen two patients recover after drainage of an accompanying empyema. It is doubtful if the case reported by Benson and Penberthy[2] should be included, since an esophageal ulcer was seen by esophagoscopy a few hours before rupture occurred. Clinically this case did present the same problems that would have resulted from a spontaneous rupture.

Vinson[9-11] has reported a number of cases of esophageal stricture that he felt were due to small spontaneous ruptures resulting in strictures upon healing. There is considerable justification in assuming that these strictures did form in this manner. Conceivably a number of these might have been due to incomplete ruptures.

A 42-year-old, well-developed, rather obese white male was admitted to the Herman Kiefer Hospital, Detroit, Mich., July 9, 1941. He appeared very ill. He was dyspneic at rest and noticeably cyanotic. His temperature was 102° F., respirations 34 per minute, and his pulse rate was 120 per minute and of poor quality. At the time of his entry the patient was too ill to give a satisfactory history. He had been admitted to another hospital four days earlier complaining of shortness of breath, upper abdominal pain, and pain in the left side of his chest. He had been treated with oxygen and sedatives and kept under observation. A chest roentgenogram had revealed a left pleural effusion. A thoracentesis had been performed, disclosing the presence of clear yellow fluid with no demonstrable organisms. He had then been transferred to Herman Kiefer Hospital with a presumptive diagnosis of tuberculous pleuritis with effusion.

Examination upon entry revealed a very sick man with the physical findings of a left hydropneumothorax. This was confirmed by fluoroscopy and x-ray. The patient's abdomen

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was distended and the presence of mild epigastric tenderness was noted. A left thoracentesis was performed immediately, and 800 c.c. of very foul smelling, thin, purulent, brownish fluid were removed. The removal of this fluid gave the patient but scant relief from his dyspnea and discomfort. Bacteriologic examination of the fluid revealed the presence of *Staphylococcus albus, Streptococcus viridans,* and *Bacillus influenzae.* No acid-fast organisms were found. The patient's white blood count was 19,800 per cubic millimeter with 73 per cent neutrophiles, of which 20 per cent were nonsegmented. His hemoglobin measured 74 per cent and his erythrocyte count was 3,700,000 per cubic millimeter. His urinalysis was essentially normal.

The patient was placed in an oxygen tent and given sulfathiazole by mouth. While in the oxygen tent he was fairly comfortable but became cyanotic if the tent was removed. The next day another thoracentesis removed 500 c.c. of the same type of fluid, again without giving the patient any relief.

Believing the patient had an acute putrid empyema probably secondary to a small spontaneous bronchopleural fistula of unknown etiology, a closed intercostal drainage of the left pleural cavity was performed, using 1 per cent procaine as local anesthetic. The intercostal catheter was connected to a water-tap suction maintaining a constant negative pressure of 20 to 24 cm. of water. This catheter failed to function satisfactorily because it frequently became plugged in spite of irrigations every few hours. Although the oral fluid intake measured 4,000 to 5,000 c.c. per twenty-four hours, the patient's twenty-four-hour urinary output was never more than 800 c.c. Usually it measured from 450 to 600 c.c. Clinically the patient gradually improved.

On July 29 an open thoracotomy by rib resection was performed by Dr. Norman J. Wilson, resident surgeon. A few hours later the dressings were saturated with a thin greenish material that had an odor much like fresh vomitus. While the dressings were being changed the patient drank a glass of water. Immediately the water gushed out of the thoracotomy wound. It was then obvious that the patient had a perforation of the esophagus with an esophagopleural fistula.

At this time a more complete history was obtained. The patient admitted to drinking an average of twenty to thirty bottles of beer daily, but stated that he rarely drank anything stronger. He had been subject to rare mild episodes of pyrosis that never inconvenienced him. He had noticed a slight nonproductive morning cough for ten or fifteen years.

On the evening of July 3 he had drunk several strong gin drinks, after which he became nauseated and vomited twice. Both times the vomitus contained fresh blood. He immediately developed a constant epigastric pain which apparently was not very severe. He soon felt ill and went to bed only to find he was dyspneic upon lying down although fairly comfortable sitting up. The next day (July 4) he drank fifteen to twenty glasses of whiskey and soda. Later in the day he again felt very ill but could be comfortable only in the sitting position. Early in the morning of July 5 he suddenly became extremely dyspneic and began to have increasingly severe pain in the epigastrium and the left side of his thorax. He then called a physician who sent him to a hospital.

As soon as the diagnosis of rupture of the esophagus was made, all food and fluid by mouth was discontinued and he was given only parenteral fluids. The advisability of doing a gastrostomy or inserting a nasal feeding tube was discussed but it was decided to observe him for a longer period of time. He seemed to be quite comfortable and continued to improve. Four days later (August 2) he vomited a small amount of fresh blood and shortly afterward passed several pints of dark clotted blood per rectum. He rapidly went into shock. He was given several whole blood transfusions within a few hours and his condition improved. No further massive hemorrhages occurred. A soft rubber nasal feeding tube was passed into his stomach and hourly feedings given through this. After a week the patient objected so strenuously to the tube that it was removed. Methylene blue given by mouth still appeared on the thoracotomy dressings a few seconds after being swallowed. He was given a soft diet and although a fair percentage of the ingested food escaped from the thoracotomy wound this seemed to be lessening daily. By September 9 there was no evidence of an esophagopleurocutaneous fistula.

At this time acid-fast organisms were found in his sputum on three occasions. An overexposed roentgenogram of the chest revealed a two cm. cavity in the apex of the partially re-expanded left lung. Subsequent sputum smears and cultures failed to reveal any acid-fast bacilli. Also subsequent roentgenograms of the chest did not disclose any cavity.

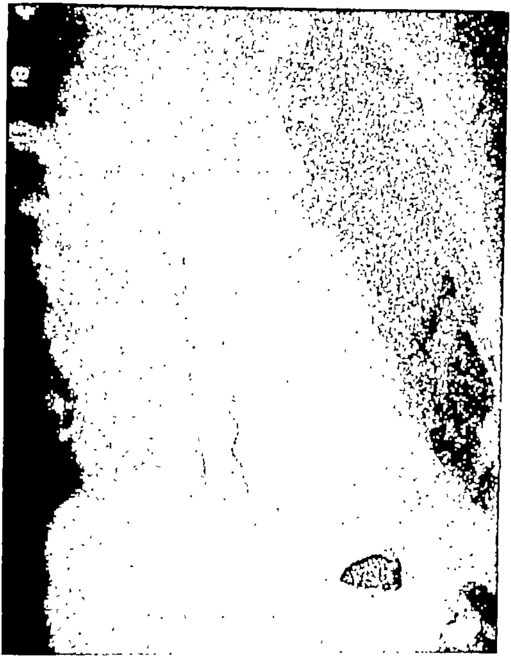

Fig. 1.—Reproduction of roentgenogram (retouched) illustrating the partial esophageal filling defect which was present after the rupture healed.

The patient continued to improve slowly and by November the empyema space was entirely obliterated. A full diet was not only tolerated very well but was demanded by the patient. On November 13, barium studies of the esophagus disclosed no stricture but there was noted a 1½ inch irregular partial filling defect on the left anterolateral wall of the distal esophagus just proximal to the cardia (Fig. 1). This was interpreted by Dr. C. O. Birkelo, radiologist, as representing scar-tissue formation secondary to the rupture. Subsequent studies five months later showed this defect to be unchanged. The patient was entirely asymptomatic at this time and has remained so. Three years later (July, 1945) he was traced and interviewed. He stated he had remained in excellent health and had no pain, dysphagia, or discomfort. It was not possible to convince him of the advisability of more x-ray studies of his esophagus.

Most commonly, the patient with a rupture of the esophagus is a male between 35 and 45 years of age who has been a heavy drinker. After an episode of forceful vomiting, with or without hematemesis, he is seized with a sudden agonizing upper abdominal or substernal pain so severe that the usual dosages of morphine give no relief. An important observation is the early but rather transient presence of subcutaneous emphysema in the neck and upper anterior chest wall. There may be considerable upper abdominal tenderness and muscular spasm. Soon there is evidence of a left hydrothorax or hydropneumothorax. Roentgenograms of the chest may reveal mediastinal emphysema or even a mediastinal fluid level if the infection remains localized to that area. Very soon, however, roentgenograms will disclose a pleural effusion, usually on the left side. The patient is acutely ill and in the course of a few hours goes into a state of vascular collapse from which he rarely recovers. The diagnosis of a spontaneous rupture of the esophagus could be quickly and easily confirmed by watching the course of a swallow of barium mixture with the patient behind the fluoroscopic screen.

While this patient did not present the classic symptoms of rupture of the esophagus, the diagnosis might have been made much sooner if the possibility of this condition had been considered. There were several details during this patient's early illness that were either overlooked or to which too little importance was ascribed. First, an adequate history of the onset of the illness was not obtained. This was an inexcusable error. Second, even though he was receiving an adequate fluid intake he was constantly thirsty, mildly dehydrated, and had an abnormally low urinary output. He received large dosages of sulfathiazole orally but his blood sulfathiazole levels were usually too low to be accurately determined. Probably most of his oral fluids and medications passed into his left pleural cavity and were removed by the intercostal suction-drainage. It was noted that the intercostal catheter became roughened and soft and that the edges of the fenestrations cut in it tended to become everted. This latter point could be seen even on one of the chest roentgenograms. This may have been due to the action of the gastric juices on the tube, although it could have been produced by the infection in the pleural space. What was thought to have been fibrin flakes blocking the tube might have been food particles or coagulated milk.

Had the pleural fluid been analyzed for the presence of gastric juice the diagnosis would have been obvious from the first. On four occasions[1, 6, 8, 14] the diagnosis of rupture of the esophagus has been made during life by this procedure.

Eliason and Welky[4] have recently reported three fatal cases of spontaneous rupture of the esophagus and have admirably reviewed the theories as to etiology and discussed the usual clinical and autopsy findings. Apparently the inciting cause is a sudden increase in intraesophageal pressure, brought on by forceful vomiting, straining, or external trauma to the abdomen or thorax. In practically all cases coming to autopsy a linear tear is found in the esophagus just proximal to the cardia. Associated with this are variable degrees of mediastinitis and left pleuritis. Sometimes the esophagus ruptures directly through the left parietal pleura where it is in intimate contact with the lower esophagus. On other occasions a mediastinitis results first with a concomitant left pleural effusion which soon becomes purulent as the mediastinal infection invades it. Whether or not pre-existing pathologic changes must be present in the esophageal wall before rupture can occur has not been satisfactorily settled.

The reason these patients die so quickly is still unexplained. Unquestionably, the sudden forceful onslaught of chemical and infective mediastinitis and pleuritis is the most important factor. Just what effect this produces on the adjacent sympathetic nerves and plexuses is largely conjecture. The patient may survive the first few days, if the infection is not overwhelming, only to succumb to a massive hemorrhage from the site of rupture when he seems to be on the road to recovery.

Because the condition is so rare and so few cases have been diagnosed during life, the literature contains but few recommendations as to therapy. Williams and Boyd[15] suggested early thoracotomy drainage. This is also suggested by Vinson.[12] Benson and Penberthy[2] reported a case of rupture of an esophageal ulcer into the pleural cavity treated by closed drainage of the empyema and the patient recovered. Eliason and Welky[4] recommend posterior mediastinotomy. This would appear to be the procedure of choice if the infection were limited to the mediastinum. If an empyema is present, the author recommends a closed intercostal drainage preferably with constant suction. This would tide the patient over his initial toxicity. When his condition had improved sufficiently and when it was felt that the mediastinum was well fixed, an open thoracotomy drainage should be done.

Surgical repair of the esophagus following spontaneous rupture has been attempted but once.[3] In this instance the patient expired twenty-one hours after operation. This is truly a formidable procedure to perform on a desperately ill patient, and, to be successful, would have to be done within a few hours after the rupture had occurred when an accurate diagnosis is most difficult. It would seem that these early hours might better be spent in attempting to resuscitate the patient by means of sedation, oxygen, parenteral fluids, and transfusions, with surgery limited to simple drainage of the infected region.

In the light of our present knowledge of chemotherapeusis the administration of penicillin in large dosages is definitely indicated. At the same time, one of the sulfonamide salts given intravenously might be a worth-while adjunct to penicillin. The efficacy of these drugs of course depends entirely upon the bacterial flora present.

Esophagoscopy is not recommended. Therapeutically it offers the patient nothing. There is real danger of further traumatizing friable inflamed esophageal tissues by its use. Observing a swallow of barium sulfate mixture under the fluoroscopic screen would give the same diagnostic aid as esophagoscopy with none of the disadvantages of the latter.

The use of a nasal feeding tube is open to strong criticism because of the possibility of producing further injury by the passage of the tube and the irritation the tube would theoretically produce by residing over the site of rupture.

Possibly these dangers are not as great as they might seem. If it is used, a chest roentgenogram should be taken immediately after passing the tube to determine its position. Vinson[13] has reported a case in which the tube was visualized looped outside of the esophagus into the mediastinum.

Whether or not a gastrostomy or jejunostomy should be done for feeding purposes is a moot question. Again this is a major operative procedure for a critically ill patient. Probably those patients who survive the first few days would recover without it if a fatal hemorrhage did not intervene. The patient could well be supported during the initial few days with fluids given intravenously and subcutaneously. As soon as the patient's condition permits, it is recommended that a jejunostomy be performed. This would place the esophagus at rest as much as is possible while at the same time providing a means of supplying much needed nutrition. This is much to be preferred to a gastrostomy, as it would obviate the danger of a reflux of food through the rent in the esophagus. In a few cases coming to autopsy it was noted that the esophageal rupture extended into the stomach. It thus seems advisable to keep the stomach as empty as possible as well as the esophagus.

It would be well to have compatible blood kept in readiness in a "bank" or refrigerator so that it might be administered with no delay if a hemorrhage occurs. The danger of this secondary hemorrhage is greatest during the first week or two. If the blood is ready and instantly available, its use may well prevent a fatality.

If the patient recovers, he should be checked at intervals of a few weeks or months by means of barium studies of the esophagus because of the possibility of subsequent stricture formation. If a stricture did occur, treatment could be started early before obstruction intervened.

With the exception of the two cases mentioned by Graham,[5] this is the first case to be reported of recovery following spontaneous rupture of the esophagus. If the entity of rupture of the esophagus is borne in mind, many more cases should be diagnosed before death. Undoubtedly the mortality rate will always be high, but if the diagnosis can be made early enough and prompt treatment begun, more and more patients should recover.

CONCLUSIONS

1. A case is presented in which the patient recovered following a spontaneous rupture of the esophagus.
2. Certain recommendations and suggestions as to diagnosis and treatment are discussed.

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HORNER'S SYNDROME AND HYPERABDUCTION NEUROVASCULAR SYNDROME FOLLOWING INTRAPLEURAL PNEUMONOLYSIS

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DENVER, COLO.

THE following case report is presented because of the occurrence in a single patient of two rare complications of intrapleural pneumonolysis.

CASE REPORT

A 39-year-old white male was admitted to the hospital on April 24, 1944, with a history of pulmonary tuberculosis discovered three years before. Roentgenograms of the chest showed extensive, exudative-fibrotic changes in the left apex and first and second costal interspaces. Phreniclasia, left, was performed on May 19, 1944. The operation was brief and uneventful. Pneumoperitoneum was induced on the following day. Following these procedures, no changes were noted in the eyes or extremities, neither postoperatively nor in a careful general re-examination on Aug. 16, 1945. The radial pulse was counted with equal ease in either wrist, both by members of the staff and by the patient himself. Beginning, however, in November, 1945, it was noted that the left radial pulse became increasingly difficult to feel, finally becoming impossible to detect. The patient had no discomfort and noted no other differences in the left arm. Artificial pneumothorax was induced on the left on March 22, 1945, and pneumoperitoneum was discontinued in October, 1945.

Because of the persistence of positive cultures of gastric washings and because of x-ray evidence of an area of uncollapsed, diseased lung held up by adhesions, an intrapleural pneumonolysis was undertaken on June 17, 1946. A broad adhesion was seen extending from the apex of the left upper lobe to the upper posterior chest wall, at the level of the first rib. Lung tissue extended to within 0.5 cm. of the chest wall. The site was injected with procaine solution through a long needle under direct observation through the cannula. The adhesion was divided, with some difficulty and some pain, close to the chest wall. In so doing, a small intercostal vessel was opened, which was subsequently sealed with the cautery. The operation lasted seventy-five minutes.

During the operation the patient's left arm was held in a position of anterolateral hyperabduction. After about twenty minutes, he began to feel numbness, paresthesias, and electric-shocklike sensations radiating down the arm to the finger tips. These became very severe but abated rapidly after the operation. The immediate postoperative course was uneventful.

On June 26, 1946, the patient experienced a feeling of pressure in the left chest and fainted. Physical and x-ray examination revealed accumulation of fluid in the left chest. Five hundred cubic centimeters of bloody fluid were aspirated, another 500 on June 29, and 400 more July 2.

Ten days after the operation the patient began to have bouts of increasingly severe pain, first in the heel of the left hand and wrist, later spreading to the volar and medial aspects of the forearm. These came on especially at night, were often associated with relative pallor and coolness of the left hand, "pins-and-needles" sensations, tremors, and local weakness. The pain became excruciating and was described as a feeling of the muscles of the forearm being "crushed and twisted." Analgesics and opiates gave only partial relief. Diathermy did not help.

A few days later it was noted that the left pupil had become much smaller than the right and the left eyelid drooped. There was a slight enophthalmos. Anihidrosis was not

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apparent. Further examination revealed absence of the subclavian, brachial, and radial pulses on the left. Blood pressure in the right arm was 120/80. On the left it was not obtainable, though fine oscillations of the mercury column were observed at 90 mm. The right palm and hand were moist, but on the left there was no sweating and the palmar skin was shiny and atrophic. Muscular weakness and some atrophy were evident in the entire left upper extremity. Repeated venous pressure studies, by the direct method, were normal on the right (5 cm. of blood), exceptionally low (2 cm. of blood) on the left. The "hand exercise test," which has been used to detect venous obstruction,[1] was negative bilaterally.

A trial injection of 30 mg. of papaverine intravenously produced no detectable change or subjective benefit. Ergotamine, 0.5 mg. intramuscularly, neither aggravated nor alleviated the symptoms. In time, the pain became somewhat localized in the lower part of the left biceps muscle. It was still very severe, and the patient was despondent. He noted that sitting up in bed and leaning on his elbows gave him a little relief, while dropping the shoulders aggravated the discomfort.

Abatement of symptoms was very gradual. By October 20 the pain and paresthesias were entirely gone, and great improvement in muscle strength was obvious. The left arm was still pulseless. The left hand appeared as warm and pink as the right, but on hyperabduction only the left quickly blanched. The palmar skin on the left was still shiny, relatively dry, and atrophic. The myosis had improved and had an intermittent character. At times the pupils were equal or the left actually larger than the right. The ptosis was slightly less marked. Enophthalmos and anhidrosis were not apparent.

DISCUSSION

The syndrome now known as Horner's was first described in 1858 by Claude Bernard, who produced it in rabbits by section of the cervical sympathetic nerves. Horner, in 1860, described a similar clinical picture in man. We have not found Horner's syndrome listed among the complications of intrapleural pneumonolysis in the textbooks. That it may follow accidental surgical injuries of the neck is well known,[2] but it has not been described as complicating intrapleural pneumonolysis, except in a few foreign communications and one report in the American literature. Hanrich, Bouvrain, and Roche[3] described four cases. They point out that nerve fibers from the ciliospinal center in the medulla pass into the anterior branches of the first three dorsal nerves and thence into the stellate ganglion and cervical sympathetic structures. Vieussen's nerve, which carries fibers innervating the iris, unites the middle and inferior cervical ganglia or the inferior cervical and first thoracic ganglia. These investigators believe that during a high intrapleural pneumonolysis the stellate ganglion, Vieussen's nerve, or communicating branches may be injured. Cavalcanti and Almeida[4] also describe two cases. They conclude that the occurrence of Horner's syndrome in their cases is explained by ascent of heat radiated from the cautery during difficult section of apical adhesions. In all cases reviewed, the syndrome was observed immediately after pneumonolysis and disappeared or subsided within a few weeks.

Kross[5] reported three cases of permanent Horner's syndrome, two following thoracoplasty with Semb apicolysis and one following an intrapleural pneumonolysis that presented no technical difficulties. The latter case was also complicated by intense subcutaneous emphysema, and the acute onset of the syndrome was at the time attributed to mechanical pressure, since "it is difficult to conceive of a direct injury to the ganglia or chain from manipulation in the pleural cavity, however near the apex that might have been.'' However, although the subcutaneous emphysema subsided, the Horner's syndrome was permanent. (The position of the adhesions sectioned in Kross' case appears to have been similar to that in our patient, in whom subcutaneous emphysema was not, of course, a factor.) Kross raises the question, since the same technique was employed in approximately 150 other patients without complication, ''whether in these three instances, the apparently injured portion of the sympathetic nervous system may not have been situated outside of its usual location and thus within the area of the operative field, subjecting itself to unavoidable trauma.'' This is perhaps the most likely explanation of all, though one that is scarcely susceptible to direct proof.

Both the French[3] and Brazilian[4] papers also reported vasomotor disturbances, viz., vasodilatation of the involved half of the face and upper extremity. Such phenomena are inconstant constituents of the Horner's syndrome and by no means comparable to the severe vasospastic reaction seen in our patient. To explain the latter we believe we must invoke another mechanism, i.e., the neurovascular syndrome produced by hyperabduction of the arms as described by Wright,[6] who discussed at length the differential diagnosis and the anatomic mechanism which produces this syndrome. Wright recommended ''that special attention be paid to the state of the pulse and complaints of paresthesias and numbness from patients whose arms are in hyperabduction on operating tables. . . .'' However, four of his five cases were the result of prolonged, habitual sleeping in the supine position with the arms hyperabducted, and the fifth patient had an injury involving the shoulder. Our patient stated that up until the time of the pneumonolysis he could sleep in almost any position and only rarely assumed a prone position with arms hyperabducted above the head. After the operation he acquired the habit of sleeping only on his back, with arms folded across his chest, or else on his right side.

Wright presented data in his paper to demonstrate that occlusion of the subclavian artery by hyperabduction of the arms is a normal phenomenon which occurs with varying ease. In our patient the radial pulse on the right can be made to disappear very easily indeed, by simple elevation of the arm to 180°. We believe that prolongation of such occlusion at the time of operation, in an arm of which the circulation was already subnormal (due to some unknown cause) contributed to the damage done.

We have found one additional instance of the hyperabduction neurovascular syndrome in the literature. Paull's[7] case was also the result of sleeping in the prone position with arms hyperabducted above the head.

SUMMARY

1. Neurologic and vascular syndromes caused by a functional mechanism (hyperabduction of the arms) in the absence of anatomic anomalies have been described by Wright[6] and by Paull.[7]

2. A case is reported following intrapleural pneumonolysis, during which the arm was held in anterolateral hyperabduction.
3. The patient also exhibited a Horner's syndrome, a rare sequela.
4. Improvement in both conditions has been spontaneous but very slow.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Twenty-Seventh Annual Meeting

May 28-30, 1947, Hotel Jefferson
St. Louis, Mo.

Program

Wednesday Morning, May 28, 1947

9:00 A.M. BUSINESS MEETING

9:30 A.M. SCIENTIFIC SESSION

1. **Dorsal Sympathetic Ganglionectomy for Intractable Asthma**
   Duane Carr and (by invitation) J. Hughes Chandler
2. **Experiences With a New Method for the Control of Intrathoracic Aneurysms**
   Osler A. Abbott
3. **Anteriovenous Fistula of the Lung**
   Herbert C. Maier and (by invitation) Aaron Himmelstein, Richard L. Riley, and Joseph J. Bunim
4. **Resection of a Coarctation of the Aorta Followed by Subclavian-Aortic Anastomosis: Report of a Case**
   Howard Bradshaw
5. **Aortic-Pulmonary Anastomosis for Pulmonary Stenosis**
   Willis J. Potts (by invitation)

Wednesday Afternoon, May 28, 1947

2:00 P.M. SCIENTIFIC SESSION

6. **Diseases of the Esophagus: Moving Picture Demonstration of Esophagoscopic Views**
   Paul H. Holinger and (by invitation) Albert H. Andrews, Jr.
7. **Surgical Management of Carcinoma of the Lower Two-Thirds of the Esophagus and Cardiac End of the Stomach**
   John W. Strieder
8. **Congenital Esophageal Atresia and Tracheoesophageal Fistula**
   Clayton G. Lyon and (by invitation) Stanley G. Johnson
9. **Esophageal Hiatal Hernias of the Short Esophagus Type: Etiologic and Therapeutic Considerations**
   A. M. Olsen (by invitation)
10. **Controlled Respiration in Transpleural Operations**
    Frederick R. Mautz, Claude S. Beck, and (by invitation) Harold F. Chase

Thursday Morning, May 29, 1947

9:00 A.M. SCIENTIFIC SESSION

11. **The Diagnosis and Operability of Bronchiogenic Carcinoma**
    John H. Gibbon, Jr., Louis H. Clerf, and (by invitation) Peter A. Herbut, and John J. DeTuerk
12. **Cancer of the Lung: Relationship of Topographic Features to the Interval and Late Results of Operation**
    Harold Neuhof and Arthur H. Aufses
13. **Carcinoma of the Lung**
    Ralph Adams
14. **The Treatment of Inoperable Bronchiogenic Carcinoma by Methyl-bis**
    Edward F. Skinner (by invitation), Duane Carr, and (by invitation) William E. Denman

15. **Nonmalignant Lesions of the Lung Simulating Bronchiogenic Carcinoma: Report of Thirty Cases**
    Lyman A. Brewer, III, and (by invitation) Wilfred M. G. Jones, and Frank S. Dolley

Thursday Afternoon, May 29, 1947

2:00 P.M. EXECUTIVE SESSION, HOTEL JEFFERSON

3:00 P.M. SCIENTIFIC SESSION

Address of the President
I. A. Bigger

16. **Pulmonary Edema: Experimental Observations on Dogs Following Acute Peripheral Blood Loss**
    Robert M. Eaton (by invitation)
17. **The Effect of Pulmonary Inflation and Deflation Upon the Maintenance of the Circulation**
    Samuel Alcott Thompson
18. **The Regeneration of Defects of the Trachea and Bronchi: An Experimental Study**
    Rollin A. Daniel, Jr.
19. **An Improved Method of Resection of Pulmonary Segments: Report of a Technique Used in Over Sixty Cases of Bronchiectasis**
    Richard H. Overholt, R. H. Betts, and (by invitation) F. M. Woods

7:00 P.M. COCKTAIL PARTY, HOTEL JEFFERSON

8:00 P.M. BANQUET, HOTEL JEFFERSON

Friday Morning, May 30, 1947

9:00 A.M. SCIENTIFIC SESSION

20. **The Place of Exploratory Thoracotomy in the Management of Intrathoracic Disease**
    John B. Grow and (by invitation) Martin L. Bradford
21. **Chronic Nonspecific Suppurative Pneumonitis**
    W. E. Adams and (by invitation) Richard Kirshner
22. **Pulmonary Resection for Chronic Abscess of the Lung**
    Robert R. Shaw and (by invitation) Donald L. Paulson
23. **Pulmonary Resection for Chronic Lung Abscess**
    Edward M. Kent and (by invitation) Frank S. Ashburn
24. **The Effect of Chemotherapeutic Agents on the Growing Tubercle**
    Robert G. Bloch and (by invitation) Kirsten Vinesland, Robert H. Ebert, and Erwin Levin
25. **Closed Intrapleural Pneumonolysis: An Analysis of 1,000 Consecutive Operations**
    J. Claude Day and (by invitation) Paul T. Chapman and E. J. O'Brien
26. **Preliminary Anterior Chondrocostectomy Combined With Closed Cavity Drainage and Posterior Thoracoplasty**
    Edward S. Welles and Joseph Gordon

Friday Afternoon, May 30, 1947

2:00 P.M. BARNES HOSPITAL

1. **Presentation of Five-Year Cures of Bronchiogenic Carcinoma**
   Evarts A. Graham
2. **Malignant Mixed Tumors of Bronchus and Malignant Pulmonary Cysts**
   Nathan A. Womack
3. **Diagnosis of Bronchiogenic Carcinoma by Sputum Examination**
   Thomas H. Burford and Hans Wandall

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CANCER

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By LAUREN V. ACKERMAN, M.D.

Pathologist to the Ellis Fischel State Cancer Hospital; Assistant Professor of Pathology, Washington University School of Medicine, St. Louis

and JUAN A. del REGATO, M.D.

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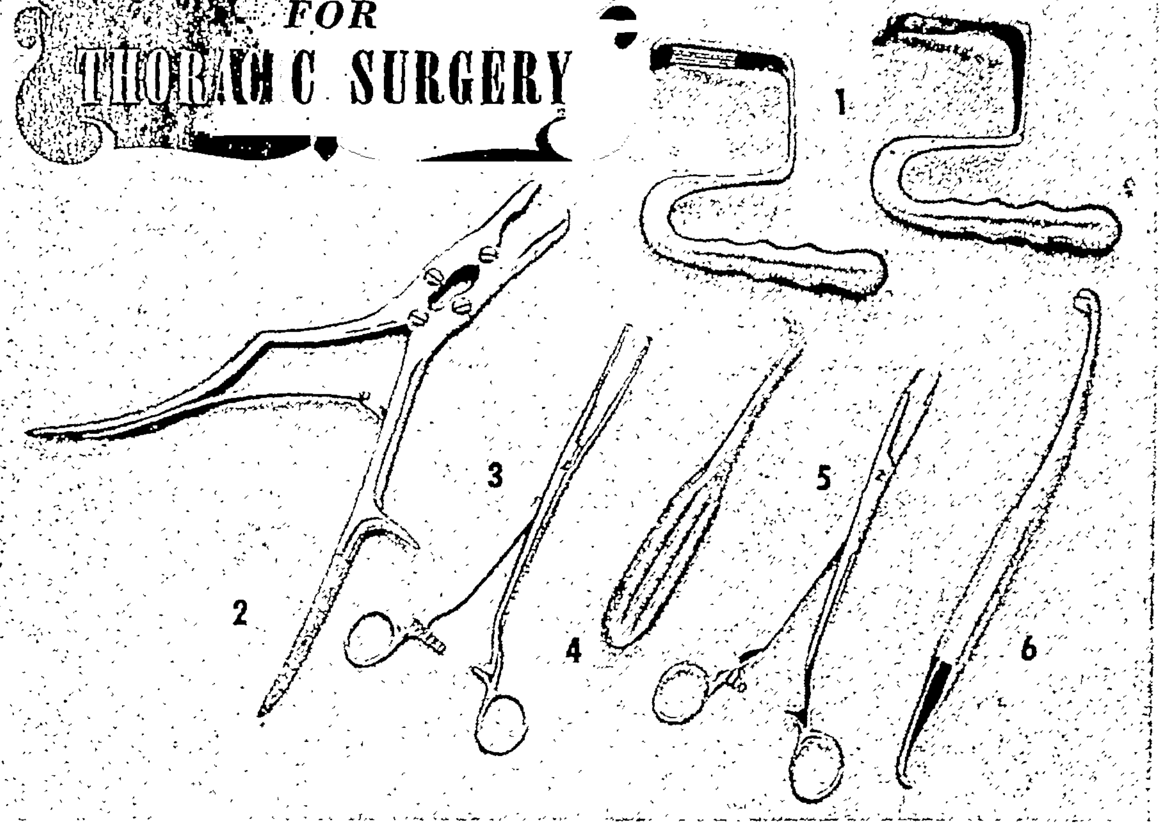

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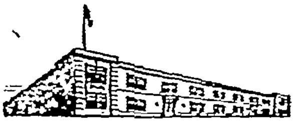

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The Journal of
Thoracic Surgery

Vol. 15 October, 1946 No. 5

Original Communications

THE DIRECT APPROACH TO CARDIOVASCULAR DISEASES

Claude S. Beck, M.D.
Cleveland, Ohio

THE importance of the group of cardiovascular diseases is shown by vital statistics of this country. The total number of deaths in 1941 was about 1.4 million. Heart disease was the cause of death in 0.4 million, which is more than one out of every four. The coronary artery problem is almost as important as the cancer problem, 107,000 deaths for coronary artery disease compared to 166,000 for cancer.

While this group of cardiovascular diseases is in first place as a cause of death it has not received equivalent funds for research. There are some interesting figures on this subject. In 1940, the amount spent per death for heart disease was 17 cents, for cancer $2.18, for infantile paralysis $525, and for other causes $4. It would appear that the amount of money available for research depends to a great extent upon public appeal. We are familiar with the "March of Dimes" and the Sister Kenny campaign. The fight against cancer and tuberculosis is always in progress. Little or no public appeal has been made to collect funds for research on the heart and blood vessels. It is highly desirable that special cardiovascular institutes be established for the study of these diseases.

Our present knowledge of these diseases concerns anatomy, physiology, pathology, and internal medicine. Until fairly recently only a small part was surgical. It is of interest to note that in the diagnosis and treatment of diseases of heart and blood vessels the internist uses methods that might be classified as indirect. These methods of study include the use of the stethoscope, the electrocardiogram, the x-ray, the fluoroscope, and such physiologic measurements as arterial and venous pressures, circulation time, temperature, etc. None of these methods involve direct vision of the part. The internist who treats heart disease seldom, if ever, sees the human heart beat. Practically no one ever thinks of this point. No one, including the internist himself, believes that here is a point worthy of comment. That is the way it has always been; that is the

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Presidential Address, read at the Twenty-sixth Annual Meeting of the American Association for Thoracic Surgery, Detroit, Mich., May 29, 30 and 31, 1946.

way it is. And yet we know that direct vision contributes something in the way of precise knowledge and understanding that cannot be obtained in any other way. We know that when a machine breaks down, a mechanic is necessary to look it over. If a car does not run, someone has to lift up the hood and look. We are now entering the era of direct examination and manipulation in the cardiovascular field. In 1905 craniotomy and laminectomy were new operations. I believe a similar development will be possible with the heart and blood vessels. It may be even a greater development because disorders of the heart and blood vessels involve mechanics and physics to a greater degree than is the case with the brain and spinal cord. It will be interesting to observe this development during the next twenty-five years.

I should like to refer briefly to the accomplishments that have been made in the direct approach to the heart and blood vessels. Many members of this association are familiar with these developments and I cannot here do more than make brief reference to them. The program of this meeting includes several contributions on the direct approach to cardiovascular diseases. These are milestones in surgical progress. These presentations are made by the individuals who made the original contributions and I dare say that this program is unique in this respect.

Let us refer briefly to the accomplishments made by the direct approach to heart and blood vessels: We begin with trauma. Methods have been described for the suture of auricular and ventricular wounds. The methods for the suture of auricular and ventricular wounds are not identical. Several excellent series of cases have been reported. Methods of procedure have been determined and are well recognized by certain surgeons, but there are many instances in which proper treatment is not given. Here is opportunity for teaching. Foreign bodies have been removed. Removal of foreign bodies from the heart is a subject that can bear further study and experimental investigation. This subject will be presented on our program. Reference should be made to the group of nonpenetrating wounds of the heart. This type of injury, I believe, is more common than the penetrating wound and better clinical recognition is in order. In the repair of vascular injuries, considerable progress has been made during the war. Small lacerations of major arteries have been repaired by suture. Excision of injured artery and end-to-end anastomosis have been done. The use of the vitallium cannula seems to enlarge the possibilities in blood vessel anastomosis. Demonstration and teaching of these methods are necessary so that surgeons generally will be able to perform these operations. No doubt, there were many instances in World War II where such opportunities were overlooked. There will be instances where they will be overlooked in civilian surgery. These methods should be taught in the experimental laboratory. The late repair of vascular injury in World War II was carried out in the vascular centers of this country. The results obtained in these centers almost reached perfection, in that there was almost no loss of life or limb in the performance of these operations.

We turn now to a group of disorders which concerns inflammation and the results of inflammation, namely fluid, adhesions, and scar tissue around the heart. This group includes purulent pericarditis, pericardial effusions, adhesive pericarditis, constrictive pericarditis, and other conditions. The term extrinsic lesions has been suggested for these conditions and the group of extrinsic lesions has been analyzed into its components which are compression, torsion, rotation, and not traction of the heart. In other words, a heart is exposed in the experimental laboratory and then an attempt is made to interfere with the function of the heart by external manipulation. The possible ways in which the heart can be disturbed by such manipulation are compression, rotation, and angulation of the heart. Let us refer briefly to one of these, namely compression. Compression may be acute or chronic as is the case with the brain. The pathology may include a variety of lesions with the heart as with the brain. There is much to recommend the adoption of this point of view. It simplifies diagnosis and it makes clear the requirements to cure the patient. For example, an almost identical clinical picture can be produced by a compression scar on the heart, by tuberculous or other type of exudate in the pericardial cavity, and by a neoplasm in the pericardium, but the treatment of these lesions is entirely different. Recognition of clinical picture is the first requirement, and determination of type of lesion is the second requirement in order to determine correct treatment. I recommend to you recognition of extrinsic lesions as a group and analysis of the various types as indicated.

We come now to patent ductus arteriosus and coarctation of the aorta. These subjects are presented on the program by those who made the original contributions. I can only compliment these surgeons for their vision and courage in doing these operations. It is not an important point to say that ligation of the patent ductus arteriosus and excision of a narrowed aorta were considered by surgeons for many years. The important event is to do the operation. This involves freeing yourself from the inertia of your associates and going ahead in experimental laboratory and with patients. I desire to refer also to the courageous step taken in ligation of the patent ductus arteriosus in the presence of infection in the blood stream. After these accomplishments are made by the surgeon there remains the problem of application of the operation in the treatment of these conditions and this is a matter of medical education. I might add that the student frequently is more apt than his teacher in learning and accepting these new developments.

Likewise the work on tetralogy of Fallot is presented on this program by those who originated the work. I think this is one of the nicest contributions made to surgery in my lifetime. It was conceived as an original idea, it was worked out in the experimental laboratory, and it was applied to patients. While it does not cure the cardiac abnormality it provides definite benefit to a condition that was hopeless. What has been accomplished in this field suggests that the direct approach of the surgeon might be extended to other cardiovascular conditions that appear hopeless.

Reference should be made to the recent developments in the treatment of portal vein obstruction. Anastomosis has been made between the splenic vein and the renal vein. The Eck fistula, of historical and experimental interest, is being applied to patients with cirrhosis of the liver. No doubt with refinements in operative skill and technique these operations will be done with greater frequency.

Resuscitation is also presented on this program. While methods of resuscitation might be improved in the future, we do possess effective methods for starting the heart from standstill and also for defibrillation of the ventricles. We possess excellent respirators for getting oxygen into the lungs. I believe courses of instruction should be provided on resuscitation so that surgeons would have in mind an order of procedure such as they have for the performance of surgical operations, instead of the disorderly procedure that usually prevails at this moment of crisis. Certification by the American Board of Surgery and the Surgical Specialties should require knowledge on resuscitation.

I cannot refer to all the advances that have been made in the direct approach to cardiovascular diseases. The prevention of pulmonary emboli by ligation of veins and by the administration of anticoagulants is a very important contribution. I can only mention sympathectomy for hypertension, for arterial spasm, for causalgia, for anginal pain, and for other conditions.

I should like to discuss briefly the coronary artery problem and show some slides of specimens that have been produced. Most of us are familiar with the trigger mechanism in the heart. A trigger is produced by reduction of arterial blood flow to any part of the heart muscle. A delicate balance may come into existence in which a small amount of oxygenated blood may prevent or promote discharge of the trigger. Discharge of the trigger produces ventricular fibrillation which is fatal. The arterial blood supply to the myocardium can be altered in several ways. One method consists of production of extracoronary anastomoses, that is, vascular communications between the coronary arteries and the arteries of tissues grafted upon the heart. Another method consists in production of a more equal distribution of arterial blood by way of the coronary arteries. Coronary arteries are essentially end arteries. Occlusion of a coronary artery or of one of its branches may produce an unequal distribution of blood to the myocardium. Communications between the peripheral branches of the two coronary arteries would produce a common arterial bed which in turn would be a protection to the heart if occlusion should develop. Such communications can be produced by surgical methods. The most effective method that I have found is by gentle abrasion of epicardium and the application of a small amount of powdered asbestos onto the abraded surface. It has been demonstrated beyond any reasonable doubt that communications produced by this method are beneficial in the presence of arterial occlusion. Another method of producing a common arterial bed in the heart is that which occurs after nonfatal occlusion of an artery. The occlusion must not destroy cardiac function because time is required for these communications to develop. I believe it is possible for the extracoronary communications and the intercoronary communications produced by inflammation to provide this time factor for these protective channels to develop. A heart with intercoronary channels is a protected heart. The specimens shown are, indeed, significant. Another method for changing or improving the coronary arterial circulation is by production of venous stasis. Experimental ligation of coronary veins does produce some measure of benefit when a coronary artery is ligated. There is also some experimental evidence to show that removal of nerves at the base of the aorta and in the region of the left coronary artery is beneficial after a major coronary artery is occluded. I have not made measurements in this subject.

It does seem to me that we have arrived at the time when some application of these data should be made to patients with coronary artery disease. I, and others working in this field, have been conservative in application to patients. It is hoped that a conservative and critical attitude will prevail in the future as further clinical application is carried out. It is hoped also that experimental studies on the coronary problem will be carried out in the research laboratories of our medical institutions. I repeat that the coronary problem is almost as important as the cancer problem and future progress in the coronary problem will be along lines of direct surgical approach.

Before concluding this address I should like to make a few remarks about possible developments of the future. Among these I would place transplantation of arteries. I have in mind excision of a segment of aorta or a segment of another important artery and replacing it by a graft taken from a less important artery. I also have in mind exclusion of the heart from the blood stream so that the heart can be opened and intracardiac operation carried out. Some progress in this field has been made. The possibilities in the way of intracardiac operations are repair of valves, removal of intracardiac tumors, removal of ball-valve thrombi, repair of defects in the auricular septum and in the ventricular septum, removal or destruction of vegetations, removal of foreign bodies, etc. These and other advances that scarcely enter one's mind, I believe, will be possible by the direct approach not only to the outside of the heart and blood vessels but to the inside of these structures as well.

SURGICALLY REMOVED FOREIGN BODY EMBOLUS IN THE PULMONARY ARTERY

Sölve Welin, M.D., Carl Axel Hamberger, M.D., and Clarence Crafoord, M.D.
Stockholm, Sweden

IT IS not unusual in wartime for foreign bodies, such as projectiles and splinters, to enter the vascular system and pass into the blood circulation. This, of course, is a much rarer occurrence in peacetime. We have nevertheless had the opportunity to diagnose and operatively remove a foreign body in the left lung which had entered it via the vascular system.

CASE REPORT

A 21-year-old apprentice at a workshop was sent to the otolaryngologic clinic from the internal medicine department with the diagnosis of foreign body in the left lung. The patient during his childhood had been perfectly healthy except for a mild attack of pneumonia at the age of 3 years. In 1943, while working in the forest, a splinter from an iron wedge struck him and entered the left groin. The accident caused such a large loss of blood that the patient lost consciousness. He was taken to a hospital in the provinces, where a lesion in the femoral vein was observed. The patient was operated upon at once. Later on an aneurysm developed, for which he was also operated upon in the autumn of 1943. Since then, he has had occasional pain and swelling in the left leg. Phlebography is reported to have shown a defective reflux from that leg.

In November, 1944, the patient caught cold and had a cough; on December 1 he had an acute attack of ague, a general feeling of illness and a stitch in the left side of the back. The patient was sent to the internal medicine department with the diagnosis of incipient pneumonia.

As a result of an x-ray examination on the following day, a small bronchopneumonic-like induration with atelectasis was observed at the base of the left inferior lobe and behind the heart. In the bronchus situated in the posterior basal part of the left inferior lobe, about 7 cm. distal to the carina, a very dense, irregularly shaped foreign body, nearly 1 cm. in length, over 1 mm. in thickness and from 7 to 8 mm. in breadth, was also revealed. It was of such density that it seemed to be a metal splinter, or perhaps, though with less probability, a fragment of compact bone (Fig. 1).

The presence of a foreign body in the left bronchial tree having thus been indicated, bronchoscopy was carried out. The mucous membrane in the trachea was found to be normal, whereas the mucosa in the left main bronchus was reddened and swollen. This was especially true at the opening of the lateral bronchial branch.

During the screening, a sound and extractor were inserted in the left bronchial branch, and one could reach to about 1 cm. from the foreign body. As there was no possibility of reaching further down, operative treatment was discontinued, it being supposed that the obstruction was due to a swelling of the bronchial mucosa.

At another x-ray examination a week later, it was found that the bronchopneumonic induration and the atelectasis had cleared. The foreign body was still in the same place as found on previous examination. It was therefore decided to make a new attempt at extraction with the aid of bronchoscopy while screening. A metal sound was inserted in the direction

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of the foreign body. On the x-ray screen, it could be seen how the sound, about 1½ cm. above the foreign body, struck a carina in a bronchial branch. If an attempt was made to push the sound further in the direction of the foreign body, a resilient obstacle was encountered; on the other hand, if one let the sound pass by, it slid medial, lateral, ventral or dorsal to the foreign body.

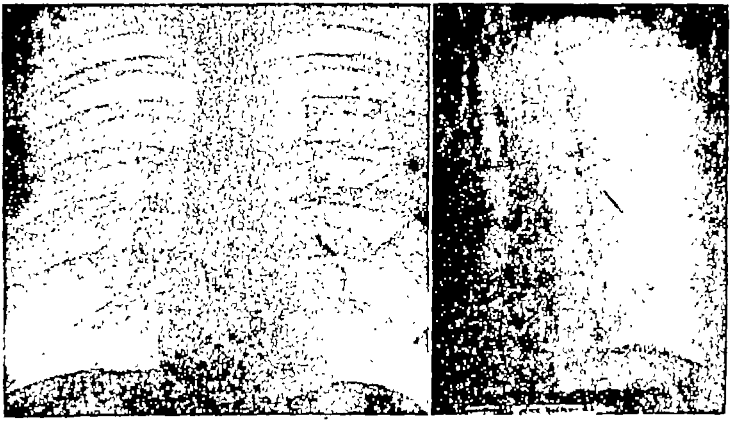

Fig. 1.—*A*, Radiogram of lung with metal splinter viewed dorsally and ventrally. *B*, Radiogram of lung in an oblique position.

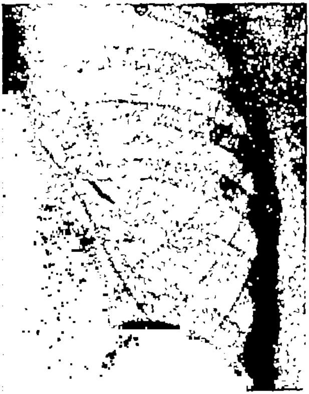

Fig. 2.—Radiogram of lung with a contrast (ureteric) catheter.

This was also the case when an attempt was made with a very small pair of tongs or a roentgen-proof ureteric catheter (Fig. 2). We therefore had the distinct impression that the foreign body could not lie in the bronchial tree and that it must be situated in the outlying lung tissue. When the bronchoscopy was planned, it was surmised that the foreign body had, on some occasion, been inhaled. The patient had, in fact, mentioned that, ten days before the illness set in, he had eaten a meat dish in which pieces of bone had been found but that it was unlikely that one of these pieces had gone the wrong way.

For this reason, not much notice was at first taken of the information that a metal splinter had entered the patient's groin. The patient, however, had mentioned that immediately after the first operation he had asked the surgeon about the metal splinter which had caused the bleeding and was told that no such object had been found during the operation. According to the patient, he was afterward x-rayed in view of finding the metal splinter, but it could not be detected.

The epicritical reasoning after the x-ray examinations and after the bronchoscopy made during screening was that the metal splinter might have entered through the groin. We know that a foreign body which has entered the venous system (for example, in a leg) will sink downward, provided the patient stands up. We also know that when a horizontal position is assumed, foreign bodies may travel upward in the venous system. In this case, the patient had been unconscious and, in view of the profuse outflow of blood, had probably been lying with his head downward. There was, therefore, a possibility that the foreign body had passed through the venous half of the heart and out into a lung artery.

After a renewed examination of the x-ray pictures, we were forced to the conclusion that the metallic foreign body was a rather thin flake with a jag on one of the edges, resembling a bit of the "barb" on the steel wedges used in forest work. Moreover, the metal piece was of such a size that presumably it could not pass into any of the fine bronchial branches situated about 7 cm. from the bifurcation.

The examination had thus indicated that, with a probability bordering on certainty, a foreign body was lying outside the bronchial tree; since its shape indicated it to be the aforesaid foreign body which had entered the patient's left groin on the occasion of the accident in 1943, the patient was sent, with the diagnosis of foreign body in the lung artery, to the Sabbatsberg Hospital for consultation with one of us (C. C.).

The patient emphasized that, so far as he could remember, there was nothing to indicate inhalation. He had not received any direct injury on the penetration of the splinter, so that the only possible way in which the foreign body could have entered was through the femoral vein in the groin. The sequelae of the accident had, however, entailed the development of an arteriovenous aneurysm. The metal splinter which had entered the femoral vein in the groin might have migrated thence to the pulmonary artery. In view of the secondary risks always involved when a foreign body lies in the lung in such a way that it may cause both vascular and bronchial erosions, it was decided to extract the foreign body before any such complications could supervene.

Operation.—The usual inclusion was made according to Crafoord, with extirpation of the sixth rib. The lung was free, without any marked changes on palpation. It was at first supposed that an indurated part, which might have developed around the metal splinter, could be felt in the hilus tract of the lower lobe, corresponding to the issue of the thickest bronchial branch. However, on passing a needle through the lung parenchyme in the induration, no metallic obstacle was encountered. The search was therefore continued, but the metal splinter could not be discovered in any other place. In order to ascertain its whereabouts, a flexible paper cassette was wrapped in sterile gloves and passed round the convex surface of the lung. A roentgen plate was then taken, with a finger being held close to the spot where it had been supposed, from the outset, that the metal splinter might be lying. The x-ray photograph confirmed our surmise. An anatomical dissection was therefore made into the hilar part of the lower lobe and the thick bronchial branch was followed up to the laterally and posteriorly situated thick artery trunk supplying the inferior lobe. When an attempt was made to loosen the artery from the bronchus within the indurated area which had been palpated, this was found impracticable, and since the artery had been exposed, it was possible, through its posterior free wall, to feel the foreign body, visible on the roentgen plate, fastened in the part of the artery wall adjacent to the bronchus. It was therefore necessary to expose the thick arterial branch central to the foreign body, as well as the thick venous branch of the inferior lobe. An artery forceps was then placed on the exposed pulmonary vein and on the artery central to the metal splinter. A forceps could not, in fact, be placed effectively peripheral to the metal splinter for it was not possible to explore all the arterial branches peripherally so that they could be clamped. The artery was then incised longitudinally, whereupon the foreign body, with relative freedom from bleeding, could be extricated from its bed in the artery wall (Fig. 3). The bed, itself, was discolored grayish black, but by careful probing with pincers, a membrane, 1 mm, in thickness, covering the whole bottom of the bed, could be loosened. When this membrane had been removed, we found a smooth grayish-white scarred surface corresponding to the place where the foreign body had previously been located. The maximum length of this surface was 17 mm., its maximum width 11 mm. An intima-free vascular surface corresponding to the surface of the foreign body was, of course, obtained. Heparin solution, 0.2 c.c., was therefore injected into the bottom of the bed between the bronchus and the artery. After suture of the artery and removal of the artery forceps, it could be seen that there was a free and satisfactory blood passage. Not even after the lapse of ten minutes, when the blood flow was still being controlled, was it possible to detect the slightest trace of a secondary thrombosis in this region. It is, of course, very remarkable that an embolus due to a foreign body should have healed into the artery without giving rise to a secondary thrombosis. It is just as noteworthy that a secondary thrombosis should not have set in after the removal of the foreign body.

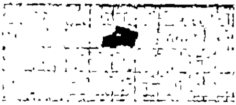

Fig. 3.—The foreign body lying on a centimeter paper.

Sulfathiazole in powder form was applied between the lobes and in the pleural cavity, and a primary suture was made according to the method of Crafoord.

After the operation, the primary suture healed without any complications. There were no clinical or roentgenologic signs, whatever, of a disturbance of the circulation in the inferior lobe of the left lung, which indicated that even in the sequel no thrombosis developed in the opened pulmonary artery.

In wartime, as already mentioned in the introduction, it is not unusual for projectiles of different kinds to enter the vascular system. Several cases in which the foreign body had entered an artery, had followed it to the periphery, and had produced symptoms resembling those of arterial embolism have been reported (Baker[1]). It is more unusual for projectiles to enter the venous system and to travel on toward the heart. Freund and Caspersohn[2] mention a patient in whom a bullet had entered the inferior vena cava and penetrated into the right ventricle, where it had become fastened in the trabeculae carneae. This patient was cured by an operation.

Henes[3] reports another case where a 31-year-old man was hit by a grenade splinter which passed through the lung into the inferior vena cava. The patient seemed fairly well for the first few hours but gradually bled to death. The autopsy showed a large hole in the extrapericardial part of the inferior vena cava. The splinter could not be detected in the vena cava, nor in the heart, in which the endocardium and the valves were perfectly intact. It was in the right lung, in a branch of the pulmonary artery, that the splinter was eventually found wedged. The splinter, which was 2½ cm. in length, triangular with sharp edges, passed through the chest wall and right lung, entered, with reduced force, the inferior vena cava, and then followed the blood stream through the heart and out into the lung. The curious thing is that this rather big splinter had not in any way injured the heart. The author stated that the time taken by the foreign body in traveling this long way must certainly have been very short. The splinter had presumably passed the heart before the patient was admitted to the hospital. No heart trouble had, in fact, been observed during his stay there.

Paltauf[6] reports a case in which a shot had pierced the lung, passed into the pericardium and continued into the pulmonary artery. The projectile, which was 6 by 35 mm., was found in the right lung. The corresponding part of the lung showed hemorrhagic infarction and the pleura showed fibrin deposits. The patient died after seven days. The autopsy showed that the projectile had been thrown into the pulmonary artery much in the same manner as an embolus. The curious thing in this case was that the patient should have lived as many as seven days. On admission to the hospital, the heart beat was not abnormally rapid. The pulse was regular and strong. On the fourth day after the accident, a hemothorax with 650 c.c. of blood was found by aspirating. The sudden death on the seventh day was believed to have been caused by a hemorrhage into the pericardium. It is surprising that the bleeding should have been so long delayed. It may have been started by the parting of the vascular walls, which had previously been adherent. Possibly the hemorrhagic infarct with pleural lesions may have provoked a cough, thus increasing the pressure in the pulmonary artery. Such foreign body emboli have been described in case reports by several authors. Paltauf thus found in the medical literature thirty-one of these cases, in only four of which, however, the foreign body had entered the pulmonary artery.

Rogers[8] describes a hearsay case in which a radium needle that had been applied on the throat of a patient was eventually found in the pulmonary artery. We are not informed as to whether the needle was removed, or as to what happened to the patient.

Foreign body emboli have been produced experimentally by inserting various metal objects in the venous system. Thus Schloffer[10] placed three or five balls, 2 mm. in diameter, in the inferior vena cava of two large rabbits. One of them died after an hour; the other was killed after the lapse of the same time. On the x-ray picture it could be seen that the balls had passed out into the lungs, and on dissection the balls were found in the larger branches of the pulmonary artery. Riethus[7] made similar experiments on dogs and rabbits but placed the foreign bodies in the jugular vein. He found that they stopped either in the right ventricle or in the pulmonary artery.

In our case, the foreign body, as already explained, had entered the femoral vein and had been thrown by the blood stream into the pulmonary artery. We have been unable to find in the medical literature any mention of a case where a foreign body had entered a vessel so far toward the periphery, as in this instance.

From a diagnostic point of view, the case is of the greatest interest. It may be gathered that the ordinary x-ray examination utterly failed to enlighten us as to the position of the foreign body in relation to the bronchial tree. Nor could bronchoscopy alone have cleared up the matter. Not until these two methods of examination were combined was it possible to locate the foreign body and ascertain that it lay outside the bronchial tree.

So far as we are aware, this is the first case where the diagnosis of foreign body embolus in the pulmonary artery has been made prior to operation and where the foreign body had afterward been extracted by thoracotomy plus arteriotomy plus vascular suture.

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PEPTIC ULCER OF THE ESOPHAGUS

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PEPTIC ulcer of the esophagus has usually been considered a rarity and to be found mainly where there is a congenitally short esophagus. It now seems nearer the truth to say that ulcer occurs where there is such a derangement of the mechanism of the cardia that acid gastric juice flows back easily into the lower end of the esophagus. The disorder which predisposes to ulceration is hernia of the stomach through the diaphragmatic hiatus into the posterior mediastinum. The hernia may be congenital or acquired, and may be reducible or irreducible at operation. Irreducibility may be caused by ulceration, fibrosis, and shortening of the esophagus.

When the lower esophagus is being constantly bathed by acid from the stomach, a well-defined series of changes is found there. First there is a recurrent acute esophagitis which passes on to a chronic esophagitis with recurrent acute ulceration. In the next stage an acute ulcer progresses to a typical chronic ulcer. The ulcer, with its surrounding induration, produces stenosis, and above this stricture there is intense superficial inflammation. Finally, a dense fibrous stenosis is produced. As in the stomach and duodenum, the picture may be complicated by perforation or bleeding. Symptoms of esophagitis and acute ulceration are mainly high epigastric pain relieved by food and alkalies, wind, heartburn, and regurgitation of stomach contents into the throat, especially in the recumbent position. This last may be so marked that if a patient bends after a meal the food will come up as easily as it went down, and with less effort. Acid regurgitation causes a feeling of dryness and heat in the throat, and a "hot pain" in the side of the neck. The main symptom of a chronic ulcer is dysphagia. It is sometimes possible to trace the pathologic changes in a detailed history, but account must be taken of the frequent association of the condition with peptic ulcers in the stomach and duodenum, and also with gall bladder disease.

During the last five years there have been in the thoracic surgical department of the General Infirmary at Leeds, thirty-seven patients with proved peptic ulceration and six with esophagitis, all associated with diaphragmatic and gastroesophageal inefficiency. The division of the cases into two categories has very little significance for, as has already been pointed out, the one is a stage in the development of the other. Acute ulceration probably occurs in all these patients, but if an ulcer has not been obvious at the time of esophagoscopy it has been recorded as esophagitis. Some of these patients, observed over a long period, have been transferred to the ulcer category when a later esophagoscopy has shown the development of peptic ulceration. A high proportion of the ulcer cases have been chronic ulcers with stenosis. A patient

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with dysphagia is more likely to be referred to a thoracic surgical department than is one with heartburn.

In a previous article[1] it was stated, "We are not satisfied that any treatment we have given has resulted in a complete cure for our patients." The *main purpose of this paper is to review the treatment to date and to indicate* what advances have been made.

TREATMENT

Assisted Medical Treatment

In the first place the treatment of a peptic ulcer of the esophagus is by the general medical measures used for ulcer of the stomach. The patient should be propped up during the period of rest in bed. This diminishes the regurgitation of acid into the lower end of the esophagus. It is recommended that meals should be adequate and infrequent, and that they should be washed down by a drink of water. These measures, combined with antacids and atropine, are enough to heal many acute ulcers. Septic foci have been blamed as important etiologic factors, and if these are obvious they should, if possible, be eliminated. I have tried direct treatment of the ulcer by cautery through the esophagoscope without any success, but where stenosis is present repeated esophagoscopic dilatations may be necessary. Stenosis may be due to surrounding induration and the piling up of granulation tissue, in which case *relief by gentle* dilatation is only temporary. Blockage of the narrowed lumen may be due to such things as raisins, peas, or fruit stones impacted in the stricture, in which case relief is more marked, or there may be narrowing due to fibrous stricture from partial healing of the ulcer; here dilatations often give a more prolonged result. In severe cases with extensive chronic ulceration, stenosis, and esophagitis, it may be necessary to combine general and local *rest with feeding through a jejunostomy.* When *this is done, the patient* should take water and antacids by mouth. The jejunostomy should be performed while the patient is on bed rest, and continued until esophagoscopy shows the ulcer to have healed. The tube should be left in the jejunum while mouth feeding is gradually restored and for some little time after the patient has been allowed out of bed. During this time it should be kept clean by *"feeds"* of clear water. When *satisfactory meals can be taken by mouth* without recurrence of symptoms, the jejunostomy may be allowed to close. On theoretical grounds a gastrostomy should not be as effective as a jejunostomy in providing rest for the ulcer, for, although it prevents the passage of food over the ulcer and the stagnation of food above it, it encourages gastric activity and the regurgitation of acid into the lower esophagus. There is no definite evidence about how *jejunostomy feeding stimulates acid secretion in* the stomach. Either of these operations should be undertaken only for the purpose of providing rest for the ulcerated esophagus. It should very rarely, if ever, be done for the relief of esophageal obstruction due to peptic ulcer since this can be overcome by esophagoscopy and dilatation of bougies. The blind passage of bougies is mentioned only to be condemned, unless indeed the services of a *skilled endoscopist cannot be obtained.*

Surgical Treatment

When these measures fail to produce relief from symptoms and when the general condition of the patient permits, surgical treatment may have to be considered. If the symptoms are mainly dyspepsia from esophagitis and acute ulceration, and there is no fibrous stenosis, the surgical attack should be directed against the herniation. If, however, dysphagia from chronic ulceration and fibrosis is present, it is unlikely that the esophagus can be lengthened enough to allow the hernia to be reduced, and it is to meet this contingency that the operation of excision of the ulcer with esophagoenterostomy has been devised.

Reduction and Radical Cure of Diaphragmatic Hernia.—

It has already been stressed that peptic ulcer of the esophagus is most commonly associated with shortening of the esophagus and sliding of the fundus of the stomach up through the diaphragmatic hiatus into the mediastinum. This may usefully be compared with a "landslide" hernia in the inguinal region and, like it, is accompanied by weakness and inefficiency of the muscular ring guarding the area. Sometimes the stomach slides up and down easily, sometimes it becomes fixed in the herniated position. The healthy esophagus is a very elastic organ and, if the efficiency of the muscle guarding the diaphragmatic hiatus is impaired, the difference in pressure between chest and abdomen combined with the elastic recoil of the longitudinal muscle of the esophagus is enough to loosen and stretch the fibrous and peritoneal attachments of the cardia and to produce herniation. It is often possible at operation to mobilize the cardia and lower end of the esophagus and so reduce the hernia below the diaphragm. The hiatus may then be narrowed by a few stitches to keep the organs in their natural position. If this can be accomplished, the esophageal mucosa may be protected from the acid secretion. Esophagitis and superficial ulceration may then heal. It has not so far been considered reasonable to attempt this operation in advanced chronic ulcer with narrowing of the esophagus and loss of its elasticity by induration and fibrosis. The operation has, however, been performed with marked relief of symptoms where the lesion has been less advanced.

Technique: A blood drip is given into the right internal saphenous vein. During the course of the operation about one pint is given. One hour before operation omnopon, gr. $\frac{1}{3}$, and scopolamine, gr. $\frac{1}{150}$, are given. The mouth, nasopharynx, and pyriform fossae are painted with 2 per cent amethocaine hydrochloride. Sodium pentothal is then injected into the blood drip until relaxation of the jaw occurs. An esophagoscope is passed and all retained fluid aspirated from the esophagus. By means of a spray blower, a light coat of penicillin powder is applied to the esophageal mucosa and the instrument is slowly withdrawn. A Tampax pack is passed through the esophagoscope and left in position immediately below the cricoid cartilage. Light gas and oxygen are then administered through a closely fitting face mask. In this way the danger of reflux of blood or septic fluid from the esophagus into the pharynx and trachea is prevented and the incidence of chest complications minimized. The patient is turned on to his right side and a left-sided spinal anesthetic of light percaine is given. This combination of anesthetic agents has proved most valuable in the operative approach to the esophagus. The spinal anesthetic prevents straining, while the gas and oxygen administered through the face piece maintain lung inflation and blood oxygenation at any desired level. The patient should be lying on his right side and leaning neither forward nor backward so that access to the abdomen and the posterior mediastinum is equally easy. A small amount of local anesthetic is injected into the muscles of the chest wall in the region of the eighth and ninth ribs so that the muscles can be cut with the diathermy and bleeding points coagulated without stimulating their contraction. An incision is made through the skin with an ordinary knife over the whole length of the ninth rib, passing forward on to the abdomen beyond the costal margin and curving upward a little behind and over the eighth rib. The whole of the ninth rib and the posterior end of the eighth are resected. The ninth costal cartilage is removed. The eighth intercostal nerve is divided and the corresponding intercostal vessels ligatured and divided. The left side of the chest is opened through the bed of the ninth rib and the rib margins gently separated. Any adhesions to the lower lobe are divided. The left splanchnic nerves on the posterior chest wall are infiltrated with local anesthetic, and 10 c.c. of local anesthetic are also injected into the posterior mediastinum above the site of the lesion. The left phrenic nerve is crushed where it passes from the pericardium to the diaphragm. The diaphragm is opened at the anterior end of the incision and this opening is enlarged toward the hiatus, but not through the muscle ring. The relation of the structures to the muscle ring can then be determined and by gentle traction on the stomach the reducibility of the hernia can be investigated. The posterior mediastinum is then opened, and the lower end of the esophagus isolated. Both vagus nerves are divided and any vessels entering the esophagus in its lower two to three inches are divided between ligatures. The reflection of peritoneum on to the stomach is cut, and the stomach can then be drawn down into its normal position in the abdomen. The muscle edges of the diaphragm are carefully cleaned. Two silk stitches are inserted behind the esophagus joining the left and right crural fibers. These are placed in such a way that the esophagus lies snugly in the opening without compression. The incision in the diaphragm is sutured, and the chest is closed in layers with an underwater drain left in for forty-eight hours.

CASE 1.—A. R., a woman aged 39 years, had esophagitis with "short esophagus."

History.—For two years the patient had pain under the left breast one-half hour after taking food, radiating into the back between the scapulae. Attacks of pain lasted about one month, with corresponding intervals of freedom. Vomiting was mainly at night and first thing in the morning.

X-ray examination showed a short esophagus with hernia of the fundus of the stomach into the posterior mediastinum. Esophagoscopy showed esophagitis of the lower two inches of the esophagus. No ulcer was seen. The opening between esophagus and stomach was widely patent.

At operation, Nov. 2, 1943, the junction of the esophagus and stomach was found in the posterior mediastinum three-fourths inch above the diaphragmatic hiatus. The lower end of the esophagus and upper end of the stomach were isolated, and the stomach could then be replaced in the abdomen with ease. A few stitches were inserted through the muscular ring of the hiatus behind the esophagus.

Since operation the patient has had no indigestion, and has gained twenty-eight pounds in weight. X-ray examination shows the stomach in its normal position.

CASE 2.—H. H., a woman aged 55 years, had esophagitis with acute ulceration and short esophagus.

History.—The patient vomited food almost every day for twelve years. There was a sensation of fullness and "boiling" in the chest followed by a burning over the lower left side of the chest and the vomiting of undigested food. Food was regurgitated into the mouth when the patient lay down. Burning sensation and pain were relieved by alkalies.

X-ray showed a short esophagus. No cardia was evident. The esophagus and thoracic stomach maintained a constant outline, giving the appearance of a dilated esophagus. Regurgitation occurred frequently. The esophagus was emptied by gravity. There was no control of regurgitation and the appearances suggested chronic dilatation of the cardia. Peristalsis in the esophagus was not seen. Esophagoscopy revealed much fluid in the esophagus. At the lower end there was an intense esophagitis with leucoplakia and irregular superficial ulceration. No stenosis was present, and there was free regurgitation of gastric juice into the esophagus.

At operation, May 15, 1945, the fundus of the stomach was found to be lying in the posterior mediastinum. When the fascial connections of the esophagus and stomach were freed, the stomach could be replaced easily in its normal position in the abdomen, without tension in the esophagus. The muscle fibers of the diaphragm around the hiatus, which were attenuated, were sutured behind the esophagus.

Since operation the patient has been free from symptoms, and subsequent x-ray has shown the structures in their normal position. There is now no regurgitation of barium in any position.

Partial Esophagectomy and Esophagoenterostomy.—

Excision of the lower end of the esophagus and upper part of the stomach followed by mediastinal esophagogastrostomy is now a well-established procedure for carcinoma of the lower esophagus and fundus of the stomach. It is too early to say whether this operation will be complicated by peptic ulcer of the lower end of the esophagus in an appreciable proportion of cases, but it certainly produces the anatomic deformity of short esophagus and herniated stomach which is the recognized accompaniment of acid ulceration of the esophagus. On theoretical grounds it would seem unjustifiable to apply the operation to stenosis associated with an active ulcer and leave a patient with the deformity which produced, or predisposed him to the original ulcer. It was decided, therefore, that excision of the affected part of the esophagus was only justifiable if a means could be found for lengthening the esophagus and reducing the stomach below the diaphragm. A loop of small intestine isolated on its mesentery, according to the method of Roux and recently used extensively by Yudin, seemed to be the most practicable. The operation was first tried on a patient with carcinoma of the fundus of the stomach causing esophageal obstruction. A description of this and other similar cases is published elsewhere. Its success justified its application to the problem of peptic ulceration.

Technique: The preoperative preparation, anesthesia, and approach are the same as for the operation already described for esophagitis and short esophagus.

The diaphragm is opened at the anterior end of the incision and this is carried backward into the esophageal hiatus. Bleeding vessels in the diaphragm are stitched. The muscle fibers of the hiatus are doubly stitched before division in order to avoid retraction of blood vessels into the crus with hematoma formation. One or two catgut sutures are passed through the cut edges of the diaphragm and left long; clips applied to these sutures keep the edges of the diaphragm apart. The incision is then carried forward into the abdominal wall for about two inches from the costal margin. Double chest retractors are inserted, the anterior ones including the diaphragm in their grip. When the ribs are separated there is a good exposure of the lower left side of the chest and upper abdomen. The transverse colon and great omentum are drawn up and the first loop of jejunum found. Inspection of the mesentery and its vessels determines the point of section of the intestine. This is usually between five and ten inches from the duodenojejunal flexure. An incision is made in the mesentery from the intestine, at the point selected, downward and through the first vascular arch. It is then continued along the length of the first arch with division and ligature of the radical trunks close to the arch. The intestine is divided between clamps at the point selected and the division of the mesentery is continued until a loop of intestine is obtained which will pass easily up to the esophagus above the site of the lesion. The free end of this loop of intestine is stitched and infolded into itself so that a blind end is formed. A hole is made in the transverse mesocolon and the loop of intestine is threaded through this. The free end of jejunum left in continuity with the duodenum is implanted into the side of the isolated loop at a point chosen to avoid tension or kinking. The free edges of their mesenteries are united. The loop is covered with moist swabs and left in the bottom of the wound. The esophagus above the ulcer is isolated after division of the lateral pulmonary ligament and incision of the mediastinal pleura. Both vagus nerves are divided. Vessels entering and leaving the esophagus at this level are also divided to increase its mobility. A fully curved anastomosis clamp is applied to the esophagus at the top of the isolated portion and a crushing clamp applied immediately above the lesion. A small pack is inserted into the mediastinum behind the point of section and the esophagus is divided immediately above the crushing clamp. The ulcerated area of the esophagus is freed down to and including the cardia itself. The cardia is divided between crushing clamps and the ulcerated area of esophagus removed. The cardiac end of the stomach is sutured and infolded. The previously prepared isolated loop of intestine is then placed in the posterior mediastinum, care being taken that no twisting of the mesentery occurs. The blind end of the intestine is tucked up behind the esophagus for about one inch so that anastomosis is performed to the anterior surface of the intestine one inch below its blind end. This makes it possible to have a peritoneal covering both behind and in front of the anastomosis. An anastomosis clamp is loosely applied to the intestinal loop. The posterior muscle layer of the esophagus is sutured to the anterior aspect of the intestine and a stay suture is left long at each end. An incision is made in the intestine parallel with the stoma of the esophagus. A continuous stitch is inserted through the mucous membrane and muscular layers of the esophagus and intestine. This is carried around the circumference of the anastomosis and is supported by one or two interrupted stitches. The clamps are relaxed and withdrawn. The anterior muscle layer of the esophagus is then stitched to the anterior peritoneal and muscle layers of the intestine. The mucosal junction is thus sunk in an "inkwell" of peritoneum-covered intestine. Penicillin powder is applied to the mediastinum around the anastomosis. The intestine is fixed to the cut edge of the mediastinal pleura by one or two interrupted sutures and is also lightly fixed to the diaphragm. The diaphragm is closed. Chest and abdominal walls are closed and an underwater drainage tube is left in for twenty-four to forty-eight hours. At the end of the operation the esophageal pack, which is anchored by a stout piece of silk, is gently withdrawn and the pharynx cleaned out. If the swab is very wet or blood-stained, the esophagoscope is passed and the esophagus carefully dried out before the patient returns to his bed.

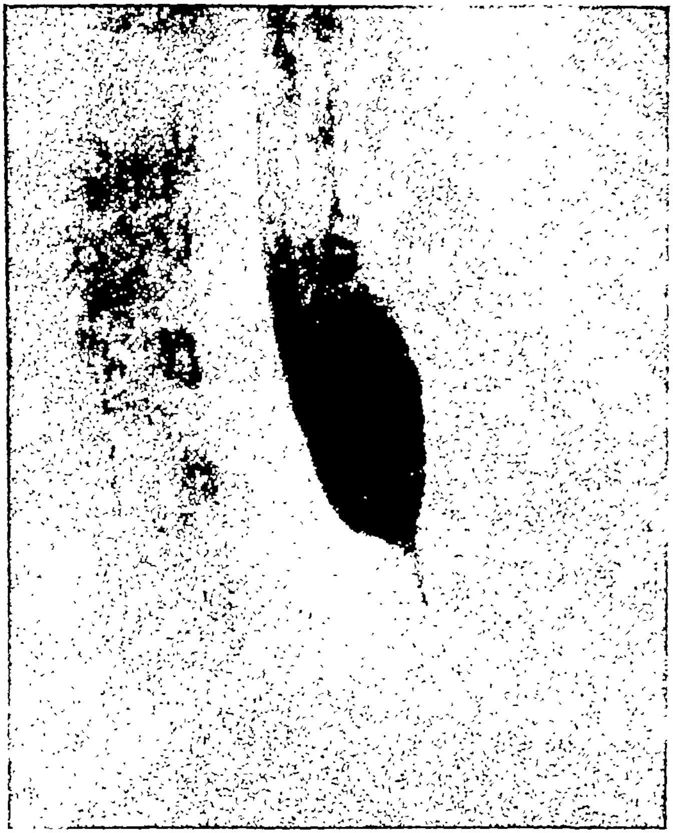

Fig. 1.—Peptic ulcer of esophagus causing complete obstruction.

Postoperative Management: The patient is nursed lying flat for twenty-four hours, during which time he is given drinks of sterile water as required. After twenty-four hours he is given glucose and fluid drinks, and penicillin lozenges to suck. After forty-eight hours the underwater drain is removed and the patient is given a high caloric fluid diet. All milk drinks are followed by a few mouthfuls of water. Aspiration of the chest may subsequently be required but is not often necessary. If performed, it should be done high enough to avoid injury to the intestinal loop, and it must be remembered that the paralysis of the diaphragm brings the loop high into the chest.

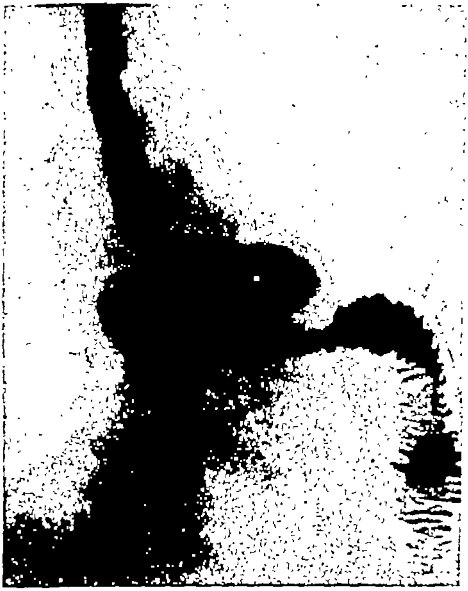

Fig. 2.—Same patient as shown in Fig. 1 after excision of chronic ulcer and mediastinal esophagoenterostomy.

Case 3.—A. McG., a man aged 40 years, had chronic peptic ulcer of the esophagus and short esophagus.

History.—He had had pain after meals for twenty years, sometimes relieved by alkalies. February, 1943, partial gastrectomy was performed for chronic gastric ulcer. This was confirmed by histology. A few weeks after this, he complained of dysphagia on swallowing solid food. Food seemed to lodge at the suprasternal notch and then gradually to go through. There was no regurgitation of food. These symptoms lasted a few weeks and then disappeared. April, 1944, dysphagia recurred. There was difficulty with fluids as well as solids. The patient lost twenty-one pounds in weight in two months.

Roentgenogram showed a smooth obstruction in the lower half of the esophagus with dilatation above (Fig. 1). The stomach could not be examined since obstruction was complete.

Esophagoscopy showed, at 34 cm. from the alveolar margin, superficial inflammation with leucoplakia and ulceration. At 37 cm. narrowing occurred. At this level the mucous membrane was covered with filmy exudate, and when this was wiped off the surface beneath was intensely red and oozing. The stenosis became complete. When a fine bougie was passed through, it dilated and the stricture was felt to be about three-fourths inch long. After dilatation the lumen gaped and was obviously surrounded by rigid inflammatory tissue. A biopsy from the ulcer showed simple chronic inflammation, with granulation tissue and fibrinopurulent exudate. The patient was kept on strict medical treatment for six months, during which time esophagoscopy was necessary on four occasions in order to relieve a complete obstruction. Since no improvement occurred, surgical treatment was decided upon.

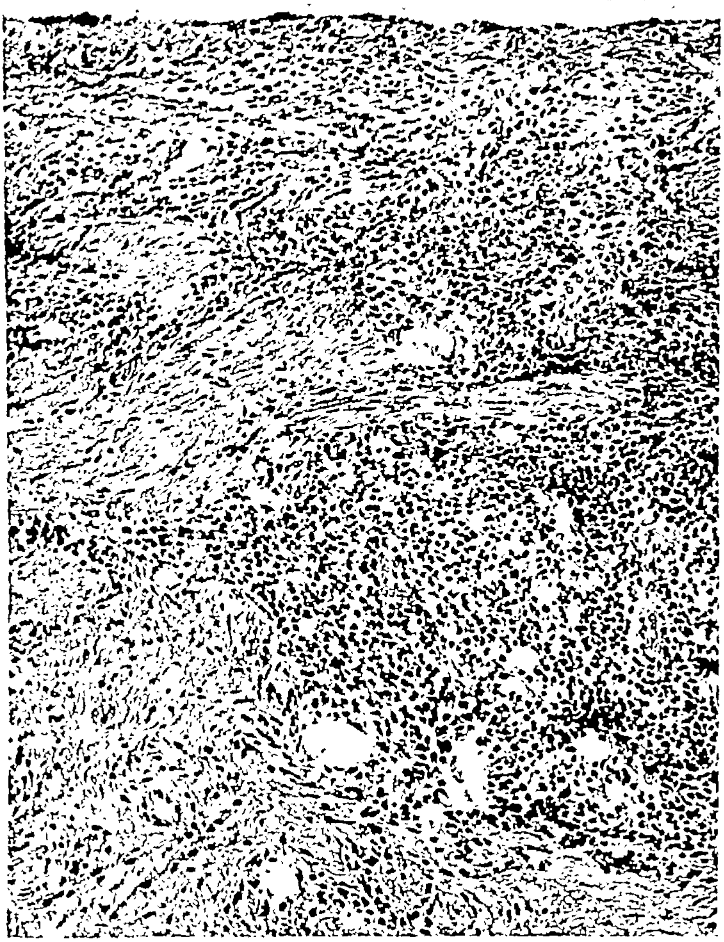

Fig. 3.—Microphotograph of chronic peptic ulcer after partial esophagectomy.

At operation, June 14, 1945, the typical appearance of herniated stomach in the mediastinum was found. The lower end of the esophagus was thickened, indurated, and inelastic. The segment of esophagus was excised according to the technique already described and a loop of jejunum was isolated, drawn up into the chest, and anastomosed to the lower end of the esophagus.

The patient has swallowed satisfactorily since. He is free from pain and vomiting. He had occasional diarrhea after meals, but since being given a nonresidue dry diet and restricted fluid intake, he has gained weight rapidly and the diarrhea has ceased. Roentgenogram (Fig. 2) shows that the intestinal loop is working satisfactorily.

The operation was slightly complicated by the previous partial gastrectomy. According to Yudin,[2] whose patients were suffering from corrosive strictures with a long expectation of life, there is no fundamental metabolic disturbance from exclusion of the stomach. It was not considered necessary, therefore, to implant the lower end of the elongated loop into the stomach. It would, of course, be easy to do this and restore intestinal continuity below, if subsequent follow-up of the patients suggests the necessity.

The excised specimen was examined* and the following report given: The specimen consisted of the lower 4.5 cm. of the esophagus, with an attached small portion of stomach wall. The lower 1.8 cm. of the esophagus seemed to be normal and covered by squamous epithelium, but a portion measuring approximately 2.7 cm. above this showed superficial ulceration. The outstanding features of this portion were (1) very great thickening of the wall (up to 1 cm. in thickness), due about equally to hypertrophy of the muscular coat and fibrous thickening of the submucosa; (2) the resulting extreme stenosis, the circumference being only about 1.1 cm. The attached portion of gastric mucosa appeared normal.

Histology.—The lesion was a chronic ulcer of the esophagus limited to the mucosa and submucosa. It was a very indolent callous-looking lesion, with dense fibrosis of the submucosa and replacement of mucous membrane by a thin layer of granulation tissue. There were scattered foci of chronic inflammatory cell infiltration (lymphocytes at the center, plasma cells and eosinophile cells at the periphery). The chronic inflammatory process only involved the muscular coat to a very slight extent. This included, however, small lymphoid nodules between the fibrous floor of the ulcer and the muscularis. There were also scattered small foci of lymphocytic and plasma cell infiltration in the substance of the muscularis. There were none of the classical layers of the gastric peptic ulcer, at least there was no zone of fibrinoid necrosis but only a thin inconspicuous more cellular surface zone, the rest of the floor being composed of dense fibrous tissue, with the focal accumulations of chronic inflammatory cells referred to previously. The ulcer would appear to be strictly limited to the esophagus. A second section taken at the gastroesophageal junction showed no evidence of peptic ulceration. Two regional lymphatic glands examined were actively hyperplastic and showed considerable anthracosis.

Diagnosis.—The diagnosis was chronic callous peptic ulcer of lower esophagus.

SUMMARY

1. Where the lower end of the organ is frequently subjected to digestion by acid gastric juice, peptic ulcer of the esophagus occurs. This happens where inefficiency of the diaphragm allows the stomach and abdominal esophagus to slide up into the mediastinum. Such herniation may be congenital or acquired.

2. Where medical and assisted medical measures fail to relieve symptoms, the length of the esophagus may be restored and the stomach replaced in the abdomen. The size of the hiatus may be diminished to maintain the organs in their normal position. Illustrative cases are recorded.

3. When chronic ulceration and fibrosis lead to inelasticity of the esophagus, reduction of the anatomic deformity may not be possible. The lower end of the esophagus may then be excised and continuity restored by a loop of small intestine on an elongated mesentery. An illustrative case is recorded.

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*Examination by Professor M. J. Stewart.

INTRATHORACIC GASTRIC CYST

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THE subject of intrathoracic gastric cysts necessitates a consideration of some of the wide variety of cysts which occur in the mediastinum. We are not concerned here with those cystic formations which originate in the lungs. If the rather common dermoid-teratoma group and the rare cystic hygromas or lymphangiomas occurring in the mediastinum are excluded, the two types of mediastinal cysts which seem to be best defined are the bronchogenic cysts and the cysts of enteric origin.[1, 2] These latter cysts have been referred to also as esophageal duplications. They possess a lining similar to that of any portion of the gastrointestinal tract, not necessarily the lining of the esophagus. Gastric cysts located in the mediastinum are proper members of the latter group of cysts.

In 1944, Ladd and Scott[3] pointed out that gastric cysts located in the mediastinum may be included best under the term "esophageal duplications and mediastinal cysts of enteric origin." They have shown that the mucosa of these gastric cysts need not necessarily be the same as that of the esophagus but may closely simulate gastric mucosa. Some of the cysts have elements of both esophageal and gastric mucous membranes.

These cysts are located usually in the posterior mediastinum at the root of the lungs and are adherent to the lungs and mediastinal structures.[4] They may be adherent to, or partially embedded in, the diaphragm or even extend below the diaphragm as in the case being reported.

The genesis of gastric cysts has been ascribed to a pinching off of a bud or diverticulum of the embryologic foregut. Ladd and Scott[3] stated that, "The ideas of Keith and Bremer offer the most rational explanation of the embryologic development and subsequent form of alimentary duplications. A duplication results from the failure of coalescence of vacuoles in the solid stage of the alimentary tube in a transverse manner. The greater the distance between the sequestrated vacuoles from the true lumen of the primitive alimentary tube, the more likelihood of there being no attachment at all between the duplication and the alimentary tube."

The intimate attachment of these cysts to the esophagus is of most importance surgically. The muscular coats are often fused to those of the esophagus or the cyst may have a common muscular wall with the esophagus. However, some of the cysts have no attachment to the esophagus. The tendency of these cysts to undergo peptic ulceration, with its ensuing complications,

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in a few cases is significant. As a result of ulceration it is possible that bronchial communication may be established rather than pulmonary compression and extrinsic bronchial obstruction which usually occur.

Intrathoracic gastric cysts usually become apparent in infancy and childhood. The symptoms are pain, cough, dyspnea, dysphagia, and regurgitation of food. Either hemoptysis, hematemesis, or both may occur. Repeated infection within the cyst may be the basis for supposed recurrent attacks which are erroneously considered to be pneumonia, but superimposed pneumonia in the lobes in close proximity to the cyst is common. Chronic pulmonary suppurative disease, including bronchiectasis, may occur in the lobes immediately surrounding the wall of the cyst. If the cyst is not attached to the esophagus, the ideal surgical treatment is removal in one stage. Rarely, it may be necessary to remove a lobe of the lung in juxtaposition with the wall of the cyst because of the irreparable damage to that portion of the lung due to infection and bronchiectasis which may be of long-standing. Often an intimate esophageal attachment makes primary resection difficult. When intimate attachment of the cyst to the esophageal wall makes separation impossible, the safest procedure is to marsupialize the cyst and to destroy the mucosa at some later date.

The present report is concerned with the successful surgical removal of a gastric cyst located in the mediastinum.

REPORT OF A CASE

A white girl, aged 10 years, registered at the Mayo Clinic on May 21, 1945. She gave a long history of repeated "pneumonic" attacks. Cough developed when she was 3 years of age. At this age she experienced two "pneumonic" episodes, one in September and one in December, 1937. A long period of cough productive of purulent sputum followed the second episode. When she was 7 years of age a long period of nausea and vomiting developed during which the temperature ranged from normal to 101° F. In 1941 she had pneumonia which first involved the upper lobe of the left lung and later involved the lower lobe.

In October, 1944, when the patient was 10 years of age, she had mumps, complicated by persistent nausea and vomiting which were thought to be a recurrence of the illness she had experienced at the age of 7. One week after the onset of the mumps a severe illness developed which was thought initially to be primary atypical pneumonia. It began with severe pleuritic pain, dyspnea, nonproductive irritating cough, palpitation, and fever. A roentgenogram of the thorax at that time revealed massive consolidation of the left lung. The patient was extremely ill, but eventually recovered under treatment consisting of a combination of penicillin and sulfonamides, two blood transfusions, and administration of adequate oxygen. This attack was followed by severe cough which was productive of purulent material and, occasionally, blood. The cough continued and was associated with occasional elevations of temperature until the time of admission to the clinic in May, 1945. A diagnosis of bronchial obstruction which probably originated extrinsically had been made.

Physical examination at the time of admission to the clinic revealed signs which were largely confined to the left side of the thorax. There were diminished resonance and tubular breathing over the left side of the thorax. Conduction of voice sounds was increased on this side, especially in the posterolateral aspects.

The roentgenogram of the thorax on May 25, 1945, showed a massive atelectasis involving at least the lower lobe of the left lung and possibly a portion of the upper lobe. There was a considerable shift of the heart and mediastinum to the left side. Bronchoscopy was performed and a great deal of purulent secretion was aspirated from the left side but no evidence of foreign body was found. However, there was a definite narrowing, especially

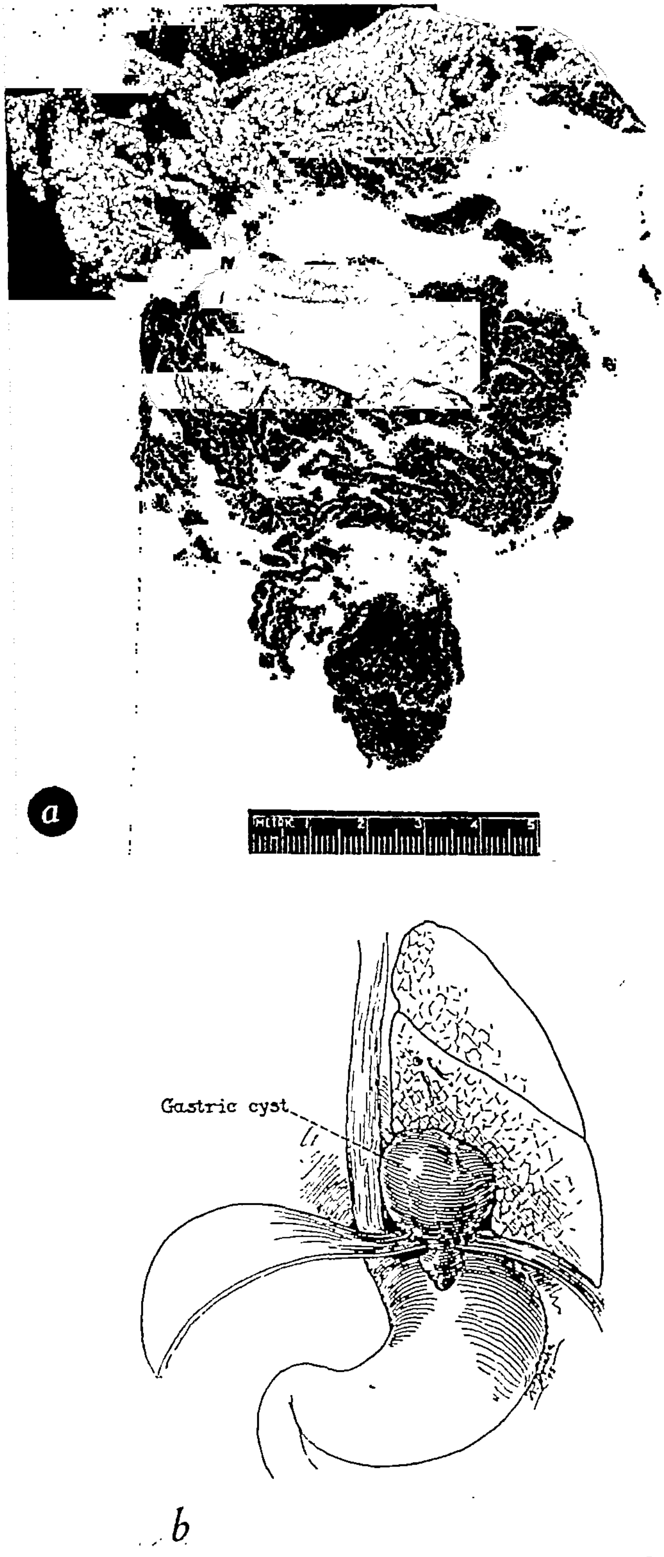

Fig. 1.—*a*, Intrathoracic gastric cyst and lower lobe of the left lung. *b*, Diagram showing position of intrathoracic gastric cyst with a portion extending below the diaphragm.

of the posterior branch of the left lower lobe bronchus. Studies in which radiopaque oil was used were made on May 29 and roentgenograms showed virtually no oil in the left lower lobe bronchus. There was atelectasis of the left lower lobe. Results of studies of the stomach and esophagus in which barium was used were negative. Examination of the blood revealed that the level of hemoglobin was 12.1 Gm. per 100 c.c.; erythrocytes numbered 6,950,000 and leucocytes, 15,000 per cubic millimeter. Results of examination of the urine were essentially negative.

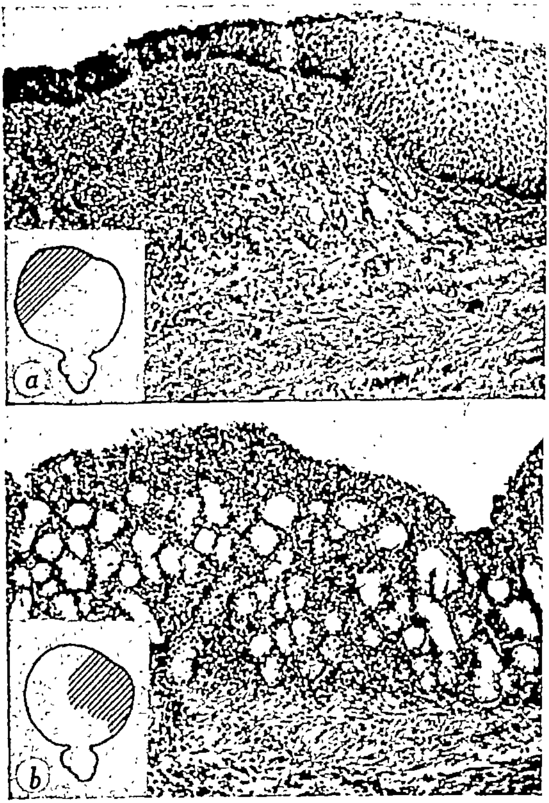

Fig. 2.—*a*, Section of squamous epithelium similar to that of the esophagus removed from the portion of the cyst wall indicated by the shaded area in the inset (hematoxylin and eosin, ×85). *b*, Portion of cyst wall as indicated lined by epithelium consisting of tubular glands lined by ordinary mucous cells (hematoxylin and eosin, ×85).

On June 7, 1945, operation was performed by one of us (O.T.C.). A primary left posterolateral incision was made, a long segment of the seventh rib was resected, and the pleura was opened. The entire surface of the left lung was densely adherent to the thoracic wall. A line of cleavage was established between the visceral and parietal pleuras and a good fissure was found between the upper and lower lobes. It was possible to carry out hilar resection with individual ligation of the hilar structures. A large cystic mass which extended down into the diaphragm posteriorly was found in the medial portion of the lower left side of the thorax. The esophagus, lying anterior to the cystic mass, was identified. The cystic mass was very adherent to the mesial and undersurface of the lung and was at first thought to be a cyst of the lung. It proved to be a congenital mediastinal gastric cyst which had become infected and established bronchial communication. Chronic suppurative disease was present in the bronchus of the lower lobe. Dissection was carried out down through the diaphragm with some difficulty. The entire cyst was removed. Temporary closed intercostal drainage was instituted and bronchoscopy was performed postoperatively.

The patient was dismissed from the clinic on June 25, 1945, in excellent general condition. The wound healed well.

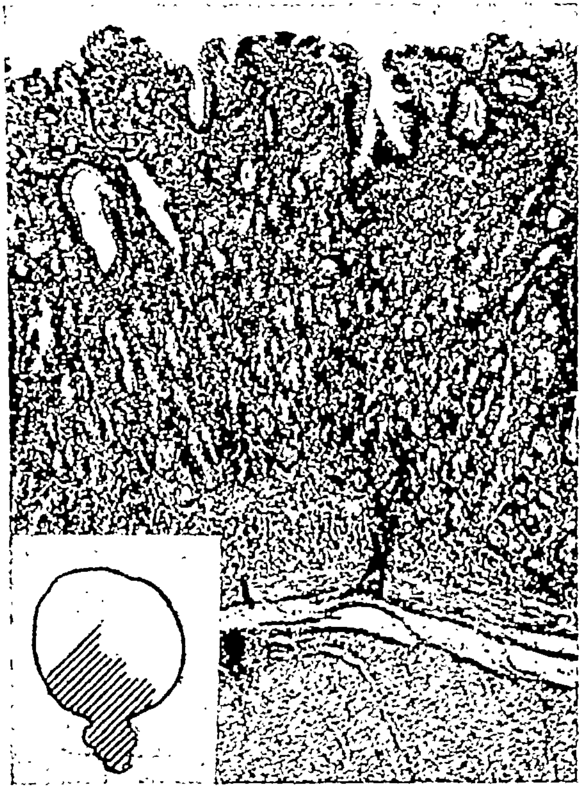

Fig. 3.—Portion of cyst wall as indicated lined by glandular epithelium containing both chief and parietal cells (hematoxylin and eosin, ×45).

Pathologic Examination.—The large, somewhat rounded sac measured 12 by 10 by 7 cm. and was rather pyriform in shape with the small lower extremity extending below the larger bulbar portion (Fig. 1*a*). Approximately seven-eighths of the sac was located above the diaphragm while the remaining portion had extended below the diaphragm (Fig. 1*b*). The sac contained thick fluid of mucoid character.

Attached to the outer surface of this lower portion of the sac was a small portion of adherent diaphragmatic muscle. The wall consisted of three distinct layers, namely, an inner epithelial layer; a middle thick muscular layer, and an outer layer which was loose, fibrous, and fatty. The epithelial layer varied in histologic appearance; it could be divided into three distinct portions. The left upper portion of the cyst was lined by stratified squamous epithelium of the esophagus (Fig. 2*a*). The right portion of the cyst consisted of tubular glands lined by ordinary mucous cells comparable to that of the pylorus (Fig. 2*b*).

The lower portion of the sac was lined by glandular epithelium with chief and parietal cells corresponding to the epithelium of the body and fundus of the stomach (Fig. 3).

There was ulceration of the lining over the left lateral surface where the sac was adherent to the lower lobe of the left lung and in this location there was a marked inflammatory reaction. The contents of the cyst were thick, mucoid, and obviously infected. The lower lobe of the left lung contained evidence of bronchiectasis and associated marked chronic pneumonitis.

From the pathologic findings this intrathoracic cyst can be considered a gastric cyst or an accessory stomach.

COMMENT AND SUMMARY

Several unusual features about this case of intrathoracic gastric cyst are worthy of consideration. The first of these was its partial extension below the diaphragm. Other gastric cysts reported have been attached to the diaphragm or embedded in it but, to our knowledge, none of the intrathoracic cysts have extended below the diaphragm. This extension was of considerable surgical importance because removal of it necessitated repair of the diaphragm following removal of the cyst.

The associated bronchiectasis in the left lower lobe was a second unusual feature. The cyst had apparently established bronchial communication. It is impossible to determine in this case what part peptic ulceration played in the establishment of such a bronchial communication. Unfortunately, studies were not carried out to determine whether the contents of the cyst contained enzymes or acid. Because of the types of mucosal linings present, including the parietal cells in sufficient quantity, and the thick, mucoid character of the contents of the cyst, it does not seem unreasonable to assume that such a cyst was producing acid and peptic enzymes.

A third feature was that the cyst contained the esophageal, as well as the gastric, type of mucosa as has been the situation in a few of the reported cases. A large number of parietal cells were found in the gastric type. The wall of the cyst was composed of three distinct layers.

There was no intimate attachment of the cyst wall to the esophagus nor did the cyst and the esophagus have a common musculature. The latter situation was important because it permitted primary removal of the cyst in one stage. This is not feasible in many cases because of the intimate attachment to the esophagus. The firm attachment of the lower lobe of the left lung to the lateral portion of the cyst wall did increase the difficulty of resection. As far as can be determined, this is the first case in which an intrathoracic gastric cyst was associated with bronchiectasis, had a portion of its wall extending below the diaphragm, and was treated successfully by removal of the cyst and lower lobe of the left lung in one stage.

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CRUSHING INJURIES OF THE CHEST

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INTRODUCTION

THE Medical Officers of the Royal Canadian Air Force did not have the opportunity of treating large numbers of chest injuries as was the privilege of the Medical Officers of the American Army attached to special chest units. However, we did see a number of crushing injuries of the chest and since our hospitals were located directly on the airdrome, we were often able to attend the patients a few minutes after the accident occurred and to follow through to the completion of their treatment.

Crushing injuries of the chest are commonly encountered in civilian practice and for the most part are not adequately treated, therefore it seems worth while to present a few cases which illustrate the necessity for early, effective treatment. carried out with an understanding of the physiology and pathology of the injured chest. The immediate concern is to keep the patient alive and if one keeps in mind the causes of death from crushing injuries of the chest and the underlying pathology, the rational treatment becomes clear.

DISTURBANCES OF CARDIORESPIRATORY FUNCTIONS

Crushing injuries of the chest may cause anoxia and death from the following disturbances of the delicately balanced cardiorespiratory function: (1) hemorrhage, (2) cardiac tamponade, (3) respiratory failure (mechanical asphyxia), (4) acute pulmonary edema.

Hemorrhage.—Hemorrhage may be from an intercostal artery, internal mammary artery, or pulmonary vessels. The signs of internal hemorrhage, pallor, shock, fluid in the chest, etc., may appear late, one hour or more after the injury, because of the tendency for intrathoracic bleeding to persist (Fig. 4, *A*). For this reason, chest injuries must be observed closely for several hours even though at first the injury does not appear severe. It is important to distinguish between hemorrhage from the chest wall and intrapulmonary hemorrhage. As long as intrapulmonary hemorrhage threatens, the lung should be kept collapsed.

Cardiac Tamponade.—Cardiac tamponade is circulatory failure due to inadequate filling of the heart resulting from pressure or traction on the auricles and superior vena cava.

It may be caused by displacement of the mediastinum from pressure pneumothorax, hemothorax, massive atelectasis, diaphragmatic hernia, emphysema or hematoma of the mediastinum, and by hemopericardium.

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Cardiac tamponade is the commonest preventable cause of death in chest wounds. When treated promptly, a moribund patient can be restored with amazing rapidity.

Respiratory Failure (Mechanical Asphyxia).—Respiratory failure is the least frequent cause of death from chest wounds, for even a small area of lung, providing it is functioning properly, will sustain life. Respiratory failure may be caused by paradoxical respiration of a shattered chest wall so that on respiration stale air flows back and forth between the crushed side and the good lung. It may also result from obstruction to the airway by blood or mediastinal emphysema, elevation of the diaphragm from extreme gastric dilatation, or a bilateral tension pneumothorax. Bilateral pneumothorax will not cause respiratory failure providing the interpleural pressures are kept negative, so that air flows into the lung during inspiration.

Acute Pulmonary Edema.—Acute pulmonary edema is caused by pressure on the pulmonary veins by mediastinal emphysema or hematoma or by shock.

PATHOLOGY

It is essential in the treatment of crushing injuries of the chest to diagnose accurately the pathologic condition present and to understand clearly its effect on the physiology of the respiration and circulation. A crushing injury of the chest may fracture numerous ribs causing paradoxical respiration from a flapping chest wall. A fractured rib may puncture the lung or blood vessels or, even in the absence of rib fractures, a sudden crushing injury may tear these structures. The following pathologic conditions should be kept in mind:

(1) airway obstructed with blood, (2) flapping chest wall, (3) hemothorax, (4) tension pneumothorax, (5) massive atelectasis, (6) mediastinal hematoma, (7) mediastinal emphysema, (8) hemopericardium, (9) diaphragmatic hernia, (10) acute gastric dilatation, (11) air embolism.

TREATMENT

Oxygen should be administered immediately at the rate of 8 to 10 L. per minute, without waiting for cyanosis to develop. The first signs of anoxia, rapid pulse, dyspnea, and restlessness, may appear sometime before cyanosis develops. An immediate portable x-ray examination should be ordered without interrupting treatment, for although the diagnosis will usually be clear on physical examination some cases will be most misleading and x-ray views will be lifesaving (Fig. 1).

Blood obstructing the airway should be removed by suction with a catheter passed through the nose into the bronchus.

A flapping chest wall with painful and paradoxical respiration can be relieved by adhesive strapping encircling the chest (Fig. 2). Morphine may be given intravenously with caution to relieve pain. Depression of the cough reflex is not a disadvantage in the early treatment but rather tends to prevent further hemorrhage.

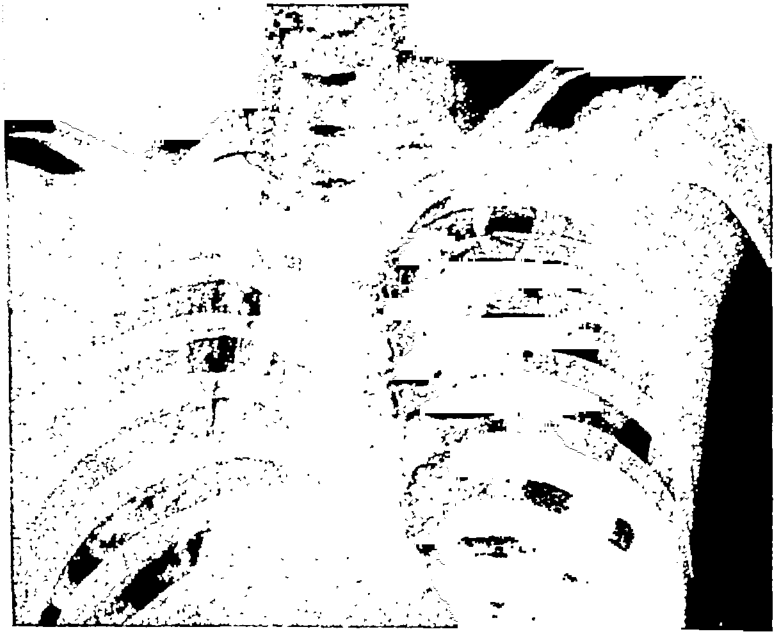


Fig. 1 (Case 1).—Post-mortem view showing massive atelectasis of right lung pulling mediastinum to right. Small pneumothorax on left. Officer was admitted to hospital fifteen minutes after aircraft crash, considered too ill for x-ray examination. Cardiac tamponade was recognized but thought due to pressure pneumothorax on the left. An immediate x-ray examination followed by a therapeutic pneumothorax on the right might have saved his life.

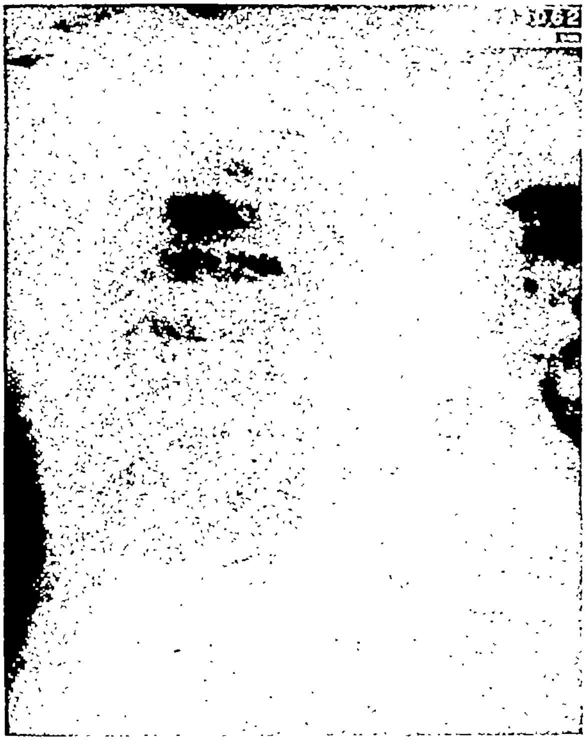


Fig. 2 (Case 2).—Multiple fractures of ribs with small traumatic pneumothorax of right side of chest. Patient was dyspneic and cyanotic from flapping chest wall. Adhesive strapping encircling the chest gave relief.

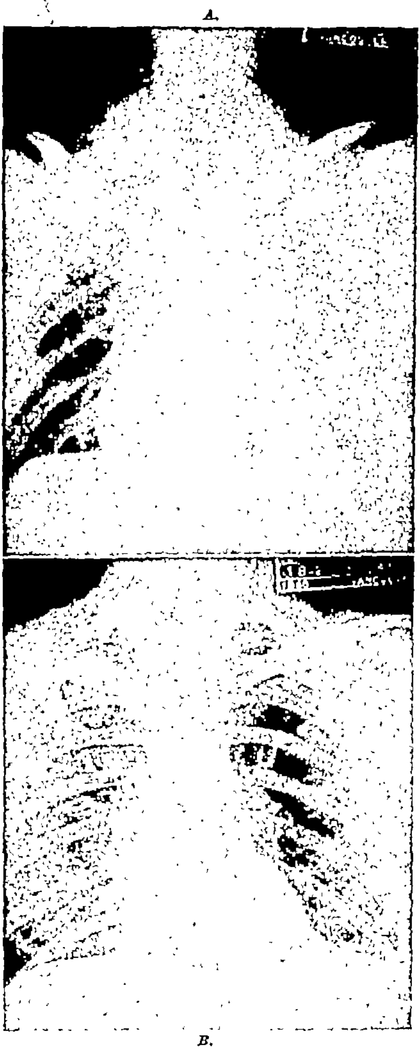
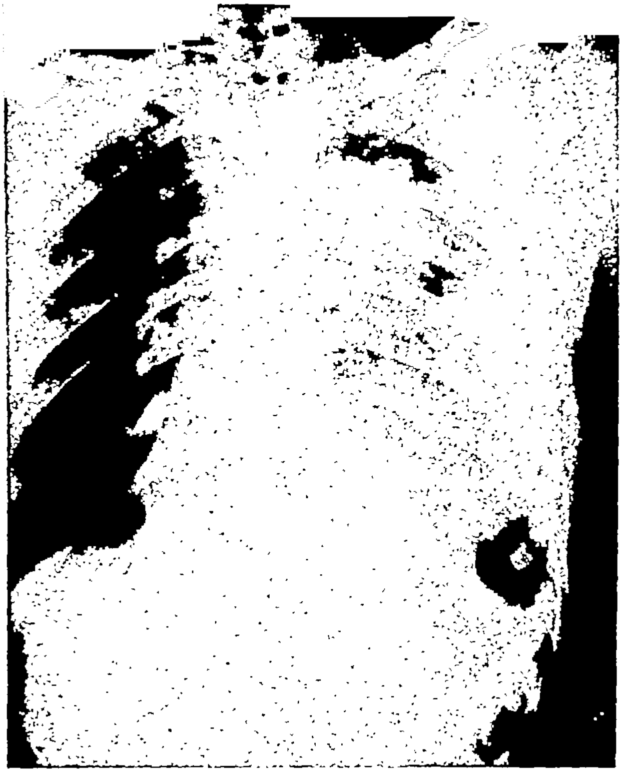


Fig. 3 (Case 3).—*A*, Hemothorax following crushing injury to left chest in car accident. Dyspnea was never severe, no cyanosis. Treated by repeated aspirations. *B*, Eight months after accident. A few adhesions to left diaphragm. Dyspnea after swimming crawl stroke 200 yards.

must be persistent, until the lung is completely and permanently re-expanded (Fig. 3, *A* and *B*). If repeated aspiration fails to remove the fluid shown to be present by x-ray examination, clotting has occurred. In the early stage it is usually possible to remove the clots with an intercostal catheter and suction of about 5 c.c. of mercury. If this is not possible, a thoracotomy is indicated to decorticate the lung permitting complete re-expansion. This should be performed as soon as the patient's condition permits before the clot has become firmly organized, usually in from two to three weeks.



A.

Fig. 5 (Case 5).—*A*, Multiple fractures of ribs and herniation of colon into left chest with moderate displacement of mediastinum to the right. Patient had been struck by a railway speeder. There was hyper-resonance with absent breath sounds in the left side of the chest. The upper part of the abdomen was tender and rigid. *B*, Barium enema reveals splenic

Cardiac Tamponade.—If cardiac tamponade is caused by tension pneumothorax, it is best relieved by the insertion of an intercostal catheter with drainage under 2 c.c. of water. The catheter should be inserted in the second interspace anteriorly to tap the dome of the pleura where the air accumulates (Fig. 4, *B*). In this way, positive pressure pneumothorax is automatically relieved without removing excess blood. When the danger of bleeding is over, usually in three to six days, the air and blood can be gradually and completely evacuated by posturing the patient (Fig. 4, *C*). The rate of evacuation is determined by the patient's symptoms. Too rapid withdrawal will cause dyspnea and a feeling of tightness in the chest. The presence of an intercostal catheter gives much better control of a tension pneumothorax. There is little tendency for the

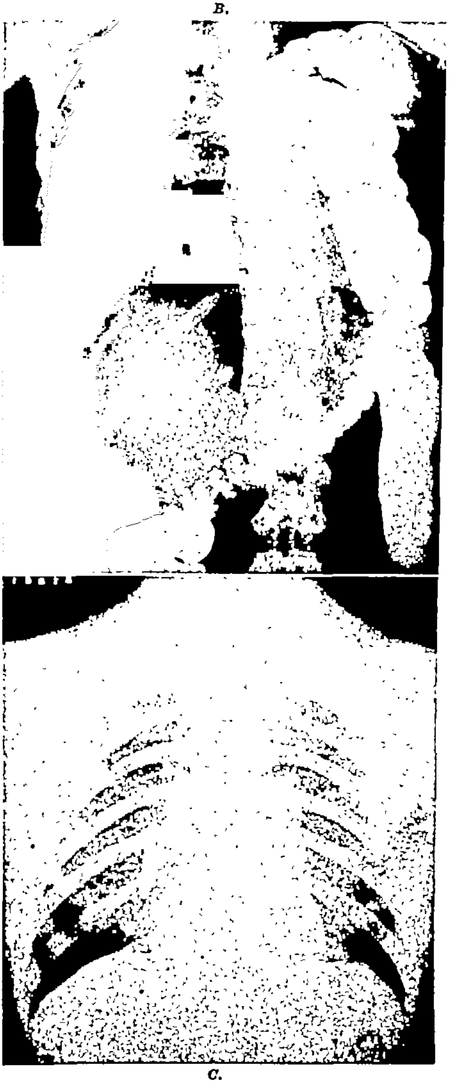


Fig. 5.—*B*, Barium enema reveals splenic flexure of colon in left side of chest confirming diagnosis of diaphragmatic hernia. *C*, Postoperative x-ray view after repair of left diaphragm from below. (Courtesy of Dr. A. B. Schinbein.)

catheter to plug, the patient is spared repeated needlings, and the chest can be strapped adequately to control paradoxical respiration. When a hemothorax is drained with an intercostal catheter, a clotted hemothorax rarely develops.

If cardiac tamponade is caused by massive atelectasis due to plugging of a main bronchus with blood clot, it will be found that a pneumothorax is not present and the symptoms will be dramatically relieved if pneumothorax is given. In addition, the hemorrhage from the pulmonary vessels will be controlled also. Later, the plugged bronchus should be aspirated with a nasal catheter or through a bronchoscope (Figs. 1 and 7).

Mediastinal hemothorax or emphysema causing acute pulmonary edema or compression of the airway is treated by an incision in the episternal notch which is kept covered with a loose petrolatum gauze dressing to allow blood and air to escape. The administration of concentrated blood serum may be of assistance. Saline solution should be avoided. Atropine, gr. $\frac{1}{100}$ to $\frac{1}{60}$, is often helpful.

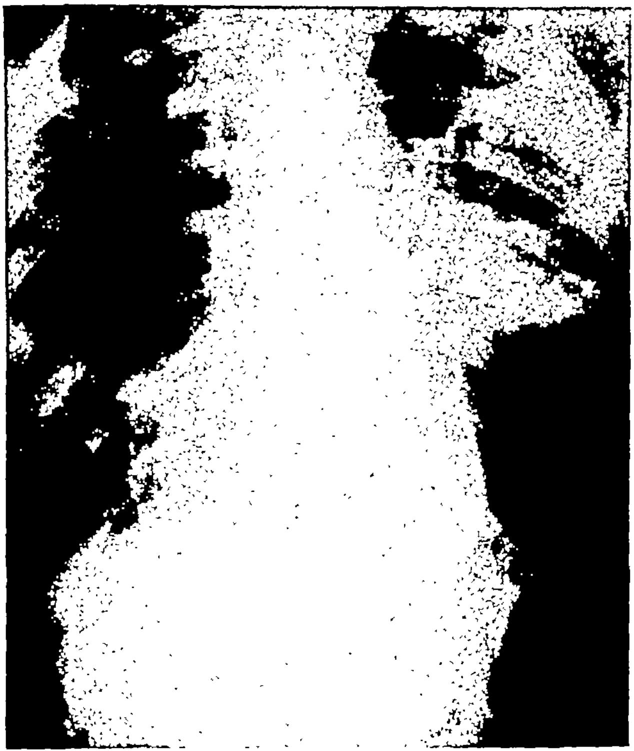


Fig. 6 (Case 6). Herniation of left diaphragm and diffuse intrapulmonary hemorrhage following crushing injury in a jeep accident. Patient was very dyspneic and cyanotic, having hemoptysis. Coarse râles were heard throughout the chest with splashing sounds at the left base. He was treated conservatively with bronchial and gastric aspiration and died eight days after injury. (Courtesy of Lieutenant Colonel Rocke Robertson.)

Acute gastric dilatation, if present, should be relieved by suction through a duodenal tube.

In one patient, air embolism occurred three days after injury while he was being moved for x-ray examination of a fractured ankle. One of the fractured ribs formed a very sharp spicule and a small traumatic pneumothorax failed to protect the lung. He had a partial hemiplegia which cleared completely in three

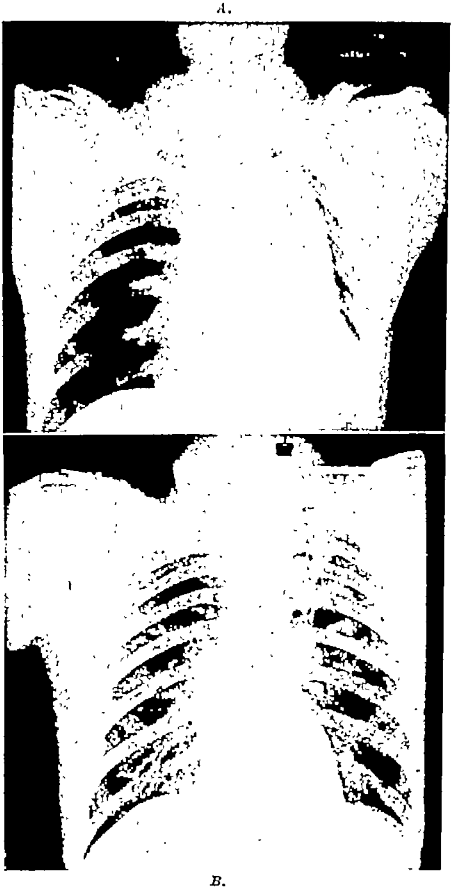


Fig. 7 (Case 7).—*A*, Massive atelectasis of left lung following hemoptysis in a patient with minimal pulmonary tuberculosis. A small left pneumothorax had been given to relieve severe dyspnea and cyanosis from pull on the mediastinum. *B*, Blood plugging left main bronchus removed five days previously by bronchoscopic aspiration. Lung now re-expanded. (From Robertson, Ross: Canad. M. A. J. 44: 111, 1941.)

weeks. This complication would have been prevented if the pneumothorax had been increased to collapse the lung out of reach of the broken ribs.

Cardiac tamponade from hemopericardium calls for aspiration of the blood from the pericardial sac.

A large herniation of abdominal viscera through a ruptured diaphragm might easily be mistaken for a traumatic pneumothorax (Fig. 5, *A*). Expansion of gases in the displaced viscera may cause acute cardiac tamponade and necessitate immediate thoracotomy for repair of the diaphragm (Fig. 6).

CONCLUSIONS

1. An intercostal catheter inserted in the second interspace anteriorly is the most effective method of controlling pressure pneumothorax and of preventing clotting hemothorax.
2. Intrapulmonary hemorrhage is best controlled by collapse of the lung by pneumothorax.
3. Bronchial secretions or hemorrhage should be removed by aspiration rather than strenuous coughing.
4. A diaphragmatic hernia is almost the only indication for immediate thoracotomy in crushing injuries of the chest.
5. In every general hospital, a team trained and equipped to treat crushing wounds of the chest would save many lives.

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DISCUSSION

DR. ALFRED BLALOCK, Baltimore, Md.—I would like to ask Dr. Robertson how he makes the diagnosis of cardiac tamponade. More specifically, I would like to know if the venous pressure was measured and, if so, what the venous pressure was; and, second, if the heart was fluoroscoped with regard to pulsations.

DR. CLAUDE S. BECK, Cleveland, Ohio.—There is a good English word to take the place of cardiac tamponade, which seems to be getting general use in the literature, and that is the simple term of ''compression of the heart.'' It seems to me that that term is better than cardiac tamponade.

DR. H. BRODIE STEPHENS, San Francisco, Calif.—It is my opinion that hemothorax is best managed by early aspiration. Early aspiration means removal of the hemothorax within twenty-four to forty-eight hours after injury. The British, under the leader-ship of A. Tudor Edwards, are particularly insistent that a fresh hemothorax be aspirated early in order to prevent a possible chronic hemothorax.

I believe it is worthy of mention to tell you of a wounded soldier I treated in a Field Hospital in the winter of 1944. This soldier had a sucking wound of the right chest due to a retained high explosive shell fragment. I débrided the sucking wound and opened the pleural cavity rather widely in accomplishing the débridement. The hemothorax present was clotted and the entrance wound on the lateral surface of the right lung near the fissure between ,the upper and lower lobes was completely dry and actually sealed. I attempted to reach the retained shell fragment by passing my finger along the tract of the missile. I could not reach the foreign body but upon withdrawing my finger from the wound in the lung, the hemorrhage was so severe that pneumonectomy was required. The resected right lung revealed that the branch of the pulmonary artery to the upper lobe had been completely severed by the offending missile.

This particular case is cited to emphasize the point that pulmonary bleeding will either cease spontaneously or result in death from exsanguination. It is my opinion that aspiration per se has little, if any, direct effect on the amount or duration of parenchymal bleeding. Early aspiration of a hemothorax brings about immediate direct relief to the cardiorespiratory system from the ill effects of fluid under pressure, and in addition such management of hemothorax may prevent so-called chronic hemothorax.

DR. ROSS ROBERTSON, Vancouver, Canada.—In reply to Dr. Blalock, we did not use the fluoroscope to diagnose cardiac tamponade. It can usually be diagnosed by physical examination, by noting the position of the heart and the trachea, but the cause of it may be difficult to diagnose, and for that reason the patient should have a good portable x-ray examination.

As to the use of the term "tamponade," I would prefer to say that there is some interference with the filling of the heart. That interference need not be due to pressure. It may be due to traction on the mediastinum. Any displacement of the mediastinum may cause it.

In reply to Dr. Stevens, about the early aspiration of hemothorax, I think that most authors have first seen these patients a day or two after their injury, and if that is what Dr. Stevens means by early aspiration, I agree heartily with him, but when a patient is seen within one hour or so after injury, when he is still expectorating fresh blood, I think the lung should be kept collapsed, and I think it is best to defer the aspiration until the intrapulmonary hemorrhage is under control.

ACUTE SUPPURATIVE MEDIASTINITIS

RALPH ADAMS, M.D.
BOSTON, MASS.

ACUTE suppurative mediastinitis has been, until recent years, a disease accompanied by a high mortality rate, and the occasional case with recovery was considered unusual. Patients in seven such cases have been treated in the last three years, with successful recovery from the mediastinitis in all instances. The methods of management of these cases are presented, in the belief that they may be of interest to the profession (Table I).

Certain points are worthy of emphasis.

The following symptoms are of assistance in making a diagnosis promptly: pain in the neck or chest on swallowing, pain in the epigastrium or shoulder, and pain on inspiration. Each of these patients had pain in one or another of the locations mentioned within one to six hours of the esophageal injury which caused the mediastinitis. Neck pain occurred only in the three patients with cervical esophageal leakage. Epigastric pain was an early symptom in the three patients in whom the perforation was in the lower third of the esophagus. One patient with a midesophageal perforation had substernal pain. Two patients in whom there was rapid extension of infection into the inferior pulmonary ligament and retropleurally over the diaphragm had shoulder pain. One of these patients had sharp pleuritic pain on inspiration. The earliest symptom in all cases was pain on swallowing, and it was noticed in some degree from the first fluids taken after injury, although variable in the severity and in the rapidity with which it developed.

The physical signs observed in the cervical region were tenderness, fullness, and edema along the course of the left sternomastoid muscle. Three of the patients with lower esophageal leakage had epigastric tenderness and spasm.

The temperature rose 1 or 2° F. within eight hours in all cases, and was accompanied by an elevation of the leucocyte count to levels ranging from 13,000 to 31,000 within twenty-four hours. A temporary period of apparent improvement and subsidence of reaction occurred in four of these cases between six and eighteen hours after injury, marked by decrease in pain and tenderness, and lowering of the temperature reading and leucocyte count. This transient but treacherous phenomenon may induce in the physician a dangerous and false impression that recovery has begun. It is thought to represent merely a delay between the body responses to the initial chemical type of injury of esophageal secretions in contact with the mediastinal tissues and, subsequently, to the infectious process itself.

Read (by title) at the Twenty-Sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

From the Department of Surgery, The Lahey Clinic.

Any patient suspected of having suffered an esophageal perforation should be promptly examined fluoroscopically and roentgenologically, with barium in the esophagus, with a radiologist in charge of the examination. Roentgenograms made with portable apparatus are seldom done under sufficient control of conditions to assure adequate study or interpretation of the findings. If a patient is sick enough to warrant a tentative diagnosis of suppurative mediastinitis secondary to esophageal leakage, it is imperative that the diagnosis be confirmed or excluded promptly. Procrastination and delay are factors more likely to allow him to become worse than is a trip to the x-ray department. The same reasoning is valid in regard to introduction of barium mixture into the esophagus. If the patient does not have a perforation, a swallow of barium will do him no harm. If a perforation is present, it may be demonstrated by extravasation of the barium mixture, and immediate drainage instituted. Other roentgenologic signs of mediastinal infection observed have been: air in the tissue planes of the neck and mediastinum, anterior displacement of the esophagus, widening of the mediastinal shadow, elevation of a diaphragm, and obliteration of the costophrenic or cardiophrenic angle.

The primary requirement in treatment is prompt establishment of dependent drainage. Infection within the mediastinum rapidly becomes widely disseminated along the tissue planes, there being little natural provision for localization or immunization in this region. Surgical drainage is indicated immediately after making the diagnosis.

Gastrostomy should always be done for purposes of fluid and [illegible]
ministration. In four of the cases, the gastrostomy was done as th[illegible]
procedure under the same intratracheal anesthesia as used for the me[illegible]
omy. Only a short time is required. The esophagus thereafter [illegible]
rest until the perforation has healed. Frequently, subsequent esoph[illegible]
tation of a stricture responsible for the perforation is desirable [illegible]
thread by the retrograde route provided through a gastrostomy [illegible]
fact, one may question the wisdom of attempting dilatation of [illegible]
stricture except with the guidance of an indwelling thread [illegible]
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passage of a thread through the esophagus. If it cam
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the esophagus can usually be traversed more safely th
drawn through, and dilatations then carried out wit
would seem reasonable for the physician to decline
of a patient with a stricture who refuses to have
gastrostomy.

Drainage of the mediastinum has been est
three hours of the time the patients were seen in c
confirmed. The site of drainage has been det
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lower and one mid-mediastinal infections were
the bed of the eighth rib posteriorly. The

TABLE I.

CASE	AGE AND SEX	ADMISSION DATE	PRECEDING DISEASE	CAUSE OF MEDIASTINITIS	LOCATION OF LEAKAGE	SYMPTOMS
1	64, M	2/24/43	Caustic stricture, 3 mo.	Esophagoscopy and dilatation	Lower third esophagus	Pain on swallowing; pain in epigastrium and right shoulder; pain on inspiration; fever
2	49, F	4/ 4/44	Cardiospasm, 6 mo.	Esophageal dilatation	Lower third esophagus	Pain on swallowing; fever 10 hr. after dilatation; 102° F., 30 hr. afterward; pain in upper abdomen
3	50, F	4/13/44	Cardiospasm and fibrous stricture, 8 yr.	Plummer bag dilatation to 15 pounds	Lower third esophagus	Pain at time of dilatation; temp. 99.8° F. next day; 101° F. in 48 hr.; pain in epigastrium with swallowing; painful breathing
4	70, F	2/ 5/46	Carcinoma of esophagus invading mediastinum, 5 mo.	Esophagoscopy and biopsy	Middle third esophagus	Pain on swallowing 6 hr. after biopsy; severe substernal and upper abdominal pain and dyspnea 8 hr. after biopsy; fever 100° F.
5	58, F	1/11/44	Bony spur cervical vertebra; diaphragmatic hernia; short esophagus	Perforation of cervical esophagus by bony spur during esophagoscopy	Cervical esophagus	Pain on swallowing; gradual onset; pain and soreness in muscles in left side of neck; slowly rising fever for 3 days
6	43, F	5/24/45	Plummer-Vinson syndrome; stenosis of cervical esophagus	Esophagoscopy and dilatation	Cervical esophagus	Pain on swallowing; pain in neck and upper chest; rising fever to 100° F. in 24 hr.
7	44, F	1/13/46	Cervical esophageal stenosis from old thyroidectomy injury; receiving periodic dilatations	Dilatation 10 days prior to entry	Cervical esophagus	Pain on swallowing in neck and upper right side of chest following dilatation; increasing for 10 days; fever to 102° F.; unable to swallow, 5 days; dyspnea, stridor, cyanosis, 4 hr.

approach the abscess on the side of most marked mediastinal widening and pleural reaction. The cervical mediastinal infections were drained by an incision along the medial border of the left sternomastoid muscle, and deepened lateral to the thyroid gland to reach the retroesophageal space. In each of the three cases of cervical mediastinitis, the infection was found to extend into the upper thorax. Therefore, a right-sided posterior mediastinotomy through the bed of the third or fourth rib was done at the same time to establish through-and-through dependent drainage. The right side was chosen to avoid interference from the aortic arch or subclavian arteries, or injury to the thoracic duct, as might occur with a left-sided approach.

SUMMARY OF CASES

SIGNS	LABORATORY DATA (WHITE BLOOD CELLS)	ROENTGENOLOGIC FINDINGS	DURATION OF SYMPTOMS BEFORE SURGERY	TREATMENT	RESULT
Spasm upper abdomen; rapid shallow breathing	20,000 in 4 hr.; 17,000 in 16 hr.; 31,000 in 30 hr.	Elevation right diaphragm; effusion right cardiophrenic angle and over right diaphragm; widened mediastinal border on right, no barium given	31 hr.	Mediastinotomy, right posterior; chemotherapy; gastrostomy; later retrograde bouginage	Recovery from mediastinitis
Tenderness, mild spasm in upper abdomen	9,000 in 30 hr.	Barium extravasation; elevated left diaphragm; obliterated left cardiophrenic and costophrenic angles	32 hr.	Gastrostomy; mediastinotomy, left posterior; chemotherapy	Recovery from mediastinitis
Tenderness, spasm left upper quadrant; diminished breath sounds at left base	15,000 in 48 hr.	No barium extravasation, obliterated left costophrenic angle; elevated left diaphragm; displaced esophagus in widened mediastinum	50 hr.	Gastrostomy; mediastinotomy, left posterior; chemotherapy	Recovery from mediastinitis
Epigastric tenderness and spasm; diminished breath sounds at left base	19,000 in 8 hr.	Barium extravasation; widened mediastinum; left pleuritis	9 hr.	Gastrostomy; left posterior mediastinotomy; chemotherapy	Recovery from mediastinitis
Tenderness, edema, induration of neck muscles	13,000 in 72 hr.; 18,000 in 96 hr.	Air in soft tissues of neck; anterior displacement of esophagus and trachea; deformity of trachea from pressure posteriorly	96 hr.	Cervical mediastinotomy, left; gastrostomy; thoracic mediastinotomy, upper right posterior; chemotherapy	Recovery from mediastinitis
Tenderness, edema, induration of left side of neck	19,000 in 24 hr.	Air in soft tissues of neck and upper thoracic mediastinum; abnormally increased diameter of space between trachea and cervical spine	30 hr.	Cervical mediastinotomy, left; gastrostomy; thoracic mediastinotomy, right upper, posterior; chemotherapy	Recovery from mediastinitis
Cyanosis, unable to swallow; tenderness, induration and crepitus in neck muscles	17,000 on admission	Barium extravasation; mass in cervical and upper thoracic mediastinum; anterior displacement of trachea; air in soft tissues of neck	10 days	Old gastrostomy opening re-established; cervical mediastinotomy, left; thoracic mediastinotomy, right upper posterior	Recovery from mediastinitis

In each of the cases, the mediastinal abscess was packed loosely with dry gauze. One catheter was placed for introduction of a constant drip of penicillin or sulfanilamide solution. Another catheter was placed on suction, with its inner end within the wound in the chest wall to remove the fluid and simplify the problem of nursing care. All patients were kept in Trendelenburg position for five days. The gauze packing was removed on the fifth day and a soft rubber tube inserted for maintenance of drainage. Irrigations were discontinued on the fifth day. The rubber tubing was gradually shortened as drainage decreased and was eventually removed when the cavity and sinus tracts were healed out to the level of the chest wall muscles.

Hydration, caloric and vitamin intake, and high circulating levels of sulfadiazine and penicillin were maintained through parenteral and gastrostomy administration. The chemotherapy was continued until after all signs of systemic reaction to infection had subsided.

SUMMARY

Acute suppurative mediastinitis caused by trauma or perforation of the esophageal wall historically has been attended by a high mortality rate.

In 1943 a plan consisting of immediate gastrostomy and extrapleural mediastinotomy and massive chemotherapy was adopted. Since then seven patients have been so treated and each has recovered.

The principles and details believed to be essential to the establishment of a prompt diagnosis and an effective regimen of treatment are described and discussed.

OPEN PNEUMONOLYSIS

An Adjunct to Pneumothorax and Closed Pneumonolysis

O. C. Brantigan, M.D. (by invitation)
Baltimore, Md.

OPEN pneumonolysis was first done by Friedrich[20] in 1908. Many workers realized the necessity of releasing pleural adhesions in order to bring about adequate pulmonary collapse in an otherwise ineffective pneumothorax. In spite of poor results and a high mortality in the past, a number of surgeons[1-5, 8, 12-16, 18, 19, 24, 32-34, 38] continued to favor open pneumonolysis.

The closed method of releasing pleural adhesions was developed by Jacobaeus and Beitr[25] in 1913 and, interestingly enough, this procedure had to prove its superiority over the open operation. The open method was preferred by several workers,[13, 18, 19] during the period when the closed operation was becoming established. By comparing only two cases, Sachs[30] attempted to show the superiority of the closed method. The closed method of intrapleural pneumonolysis became an established procedure in Europe before it did in the United States. Matson[28] assured it a lasting place in American medicine. Since closed pneumonolysis became an established and accepted operation, two schools of thought have prevailed concerning the open operation. Some writers have condemned the procedure as dangerous,[6, 7, 22, 29-31, 35] and others take pride in saying it died a deserved death. It is obvious that these statements often are not based upon experience. Other workers,[13-15, 36] particularly Alexander and Anderson,[2, 4] Dohnanyi,[16] and Hoppe,[24] have maintained that the procedure, properly used, has its place in the treatment of pulmonary tuberculosis.

Alexander, in 1925[1] and 1937,[2] reviewed all reported cases of open pneumonolysis. In 1925 he concluded, ". . . that thoracoplasty is preferable to pneumothorax which must be preceded by so risky an operation as open intrapleural pneumonolysis." When the literature is reviewed one must agree with Alexander, who wrote in 1937, ". . . almost without exception the results that were reported until a few years ago were unsatisfactory; many of the patients were made worse or died . . . ." It seems that Anderson and Alexander,[4] Carter,[13] and Hoppe[24] were the only workers who reserved open pneumonolysis for those patients in whom thoracoscopic examination revealed that the adhesions could not be released by the closed method. Alexander, in 1937 believed ". . . developments in the technic of open intrapleural pneumonolysis have made the operation a valuable one for a small group of patients." This statement is applicable again in 1946, since developments from 1937 to 1946

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From the Department of Surgery, School of Medicine, University of Maryland.

far exceed those from 1925 to 1937. Alexander appears to be the only author who has written about open intrapleural pneumonolysis over an extended period of time.

Knowledge in medicine and surgery has increased tremendously in the past ten years. The advances that favorably influence the results in open pneumonolysis are many. It is fitting to discuss a few of these, especially the advances that, when applied clinically, have eliminated most of the morbidity and mortality associated with past attempts at open pneumonolysis. Pneumothorax will not be carried out when a patient has a large tension cavity.[39] When either fibrotic or tuberculous bronchial stenosis exists, pneumothorax will not be instituted nor will it be continued when atelectasis occurs.[17] The necessity of doing open pneumonolysis is thus eliminated in this group of patients, where complications are frequent and serious under pneumothorax therapy alone. Fewer patients need open pneumonolysis because the closed release of adhesions is highly effective.[21] Thus, when a patient is selected for an open operation, no surprise or unexpected contraindications will be found when the pleural cavity is opened. The physiology of the lungs and pleura is better understood.

Eloesser[19] seemed surprised at the ability to open the pleura under local anesthesia. The patient usually will maintain an open airway when awake; under general anesthesia the intratracheal tube will assure an open airway. The anesthetists are more capable now than ten years ago. Surgical shock is better understood and therefore it is no longer a serious problem in these cases. The loss of the pneumothorax after open pneumonolysis is no longer a serious problem. Re-expansion of the lung occurs because of bronchial obstruction that results from either bronchial disease[9] or muscular spasm in the bronchioles.[12] Subcutaneous emphysema is a symptom of re-expansion of the lung and not a cause. Postoperative pneumonia can, for the most part, be prevented by the proper administration of a sulfonamide drug. It also appears that perhaps a sulfonamide drug will aid somewhat in preventing a spread of the tuberculous pulmonary disease.[11] Wound healing without suppuration and the elimination of pyogenic empyema has been enhanced greatly by the use of penicillin,[27] and is aided by adequate ascorbic acid therapy.[26] Empyema of the pleural cavity is practically eliminated by the use of sodium tetradecyl sulfate in azochloramid[10] and sulfanilamide.[37] Serous pleural effusions apparently are lessened by the instillation of sodium tetradecyl sulfate in the pleural cavity. By taking advantage of these factors, it would appear that open pneumonolysis could be a safe, dependable procedure in properly selected cases where closed pneumonolysis fails to produce an adequate pneumothorax.

Rigid indications and contraindications for open pneumonolysis were adopted in an endeavor to keep the operation free of morbidity and mortality. The following indications have been used: The patient, of course, must be a suitable candidate for pneumothorax. Thoracoscopy must be carried out before a decision is made. If possible, a closed release of the adhesions will be effected. If closed release cannot be done or if the adhesions are released incompletely, the remaining adhesions are studied with open pneumonolysis in mind. The patient is acceptable for open pneumonolysis if the adhesions are not too extensive and numerous, if the area of lung adherent to the parietal or mediastinal pleura is not greater than the surface area of the operator's palm, and if there is no evidence of pleural exudate, tubercles, or pleuritis. The patient's physical condition must be good enough to warrant the operation. Open pneumonolysis is contraindicated if the conditions are contrary to those enumerated; namely, extensive adhesions, large areas of lung adherence, pleuritis, pleural tubercles, pleural exudate, or if the patient obviously could not withstand operation. True pleural effusions will contraindicate open operation but small transient effusions will not.

When open operation is decided upon, the patient is given ample amounts of vitamins, especially daily doses of 500 mg. of ascorbic acid. Sulfadiazine is administered twenty-four to forty-eight hours preceding operation. The patient is matched for blood. Usually 500 c.c. of blood are sufficient, given at the time of operation. Local anesthesia combining a paravertebral nerve block and local infiltration of procaine was used for the first six operations. In the later operations an intratracheal inhalation anesthesia, usually ether, was used. The patient is placed in the supine position and an anterior incision through the third interspace is made. The breast is circumscribed and lifted superiorly. It is unnecessary to resect a rib. Adjacent ribs or costal cartilages are cut only if the absence of tissue resilience makes it necessary. The adhesions or areas of lung adherence to the parietal pleura are released in the extra endothoracic fascial plane. Hemostasis is obtained by immediate ligatures of silk. When the lung is freed completely from the chest wall and hemostasis is complete, 500 c.c. of sodium tetradecyl sulfate 1:500, in azochloramid, 1:3,300, together with 5 Gm. of sulfanilamide are placed in the pleural cavity. The third and fourth ribs are held together with one braided silk suture passed through the ribs and not around them. No attempt is made to close the pleura or intercostal space. The muscles are closed in layers with interrupted sutures. The subcutaneous tissue and skin likewise are closed with interrupted sutures. Silk is used throughout as the suture material. Before a dressing is applied, air is introduced into the pleural space by means of a syringe and needle.

After returning to his room the patient is given penicillin, 30,000 units every three hours. All sulfonamide drug medication is discontinued. The patient is given carbon dioxide inhalations every three hours and is turned frequently. An oxygen tent is used for the first twenty-four hours. Adequate vitamins are administered. Refills of air are given as necessary. The pneumothorax is maintained under fluoroscopic or radiographic control. Adrenalin in oil is given, if necessary, to relieve bronchiolar spasm. Aspiration of the intrapleural fluid is done when needed. The patient usually is discharged from the surgical ward in about ten to fourteen days.

From April 17, 1943, to April 5, 1946, a total of 171 patients were subjected to thoracoscopy. Closed pneumonolysis completely freed the lung from the mediastinal and parietal pleural in eighty, or 46.7 per cent. In forty-five patients, or 26.3 per cent, the freeing of the lung was incomplete, but it was thought at the time of operation to be sufficient to render adequate collapse of the lung. In this group, fifteen patients were recommended for open pneumonolysis if the collapse was found to be inadequate. Open pneumonolysis was done on only two of these. In nine, or 4.2 per cent, the lung was freed incompletely, and at the time of closed pneumonolysis it was thought that the collapse of the lung would be inadequate to control the disease. Of these nine, five were recommended for open pneumonolysis if the collapse proved inadequate, but only two were subjected to open operation. Thoracoscopy alone was done on thirty-seven, or 21.6 per cent of the original 171 patients. In this group eleven patients, or 29.8 per cent, were recommended for open pneumonolysis and each patient was subjected to the open operation. Since each patient in this group that was recommended for open pneumonolysis actually underwent the operation, there are no patients who can justly be considered as controls for the comparison of end results. In the group that had incomplete release of adhesions, there are so many variable factors that it is useless to compare the end results between those who had the open operation and those who did not. Many of the patients who had incomplete release of the adhesions by the closed method subsequently were found to have adequate pulmonary collapse.

Of 171 patients subjected to thoracoscopy, thirty-one were recommended as candidates for open pneumonolysis and fifteen of these had the operation. Of the thirty-one recommended for possible open pneumonolysis, twenty had adhesions partially released by the closed method. Only four of the twenty had the open operation. It is impossible to determine how many should actually have been subjected to the open operation. A late tuberculous empyema or a spread of disease to the contralateral lung might have been averted by completely freeing the lung. Of the thirty-one patients who had thoracoscopy, only eleven were recommended for open operation, and all of these were subjected to the open pneumonolysis. It is impossible to summarize end results on the fifteen who had open pneumonolysis, since the cases are so few and the time element so variable. The details of each case are given in Table I. The operation was successful in every instance. In all patients, with one possible exception, the disease has been controlled in the lung on the side subjected to open pneumonolysis. No morbidity, no wound infection, and no empyema occurred after the open operation. In the first few operative months spread of the disease did not occur in any patient in either the ipsilateral or contralateral lung, and no patient was made worse by the open pneumonolysis operation. Three patients now have a poor prognosis because of contralateral disease. Three others have been converted to an oleothorax because of obliterative pleuritis. Two patients have persistent fluid and in each the fluid, when cultured, revealed tubercle bacilli. One patient, who developed an obliterative pleuritis and was converted to an oleothorax for this reason, had a sputum that was negative on culture seven months postoperatively. He died twenty-one months after the operation. The circumstances of his death are unknown, but the cause of death is listed as pulmonary tuberculosis on the death certificate.

Perhaps the indications for open pneumonolysis have been held entirely too rigid. Where thoracoscopy alone was done only 29.8 per cent were recommended for open operation. It is possible that more should have been recommended. Hoppe[24] in 1937 subjected to open pneumonolysis only 7 out of 220 patients who had closed release of adhesions or examination alone. However, the rigid indications undoubtedly account for the absence of postoperative morbidity and mortality and also for the good end results. If indications are to be relaxed at all it seems that one might accept more complicated adhesions and larger areas of adherence of lung to the chest wall for operation. It does not seem feasible that one-half the pleural surface can be denuded without complications. In such cases thoracoplasty surely would be indicated rather than open pneumonolysis.

Since the experience with early thoracoscopic examinations and the release of adhesions had been not only satisfactory but highly desirable,[25] the open pneumonolysis was often accomplished within two weeks of the date that pneumothorax was initiated. This reduced pleural effusions and pleuritis as factors in refusing the open operation. When the patient is operated upon early after the initiation of pneumothorax, dense fibrous adhesions are not encountered and, of course, the pleura is delicate. The patient can thus be admitted to a general hospital, pneumothorax initiated, and thoracoscopy accomplished. If needed, an open operation can be performed and the patient discharged from the hospital in from three to four weeks with an adequately controlled pneumothorax.

At one time it was thought advisable to be prepared to follow immediately with an open pneumonolysis if the closed method failed. This was discontinued since it frequently meant unnecessary preparation of the patient and useless preparation in the operating room. Such procedure often frightened the patient when a simple closed pneumonolysis was entirely sufficient or when open pneumonolysis was found to be contraindicated after thoracoscopy. When necessary, open operation now follows thoracoscopy in a few days.

From close observation it appears that patients tolerate the administration of general anesthesia better than the local anesthesia. However, there is less difficulty with re-expansion under local anesthesia since the patient struggles on recovery from general anesthesia. The struggle that accompanies the excitement in recovery from anesthesia increases the intrapleural pressure and thus favors expansion of the lung.

The supine position with an anterior incision is used because there is less chance of aspirating bronchial secretions into the other lung. If the patient is in the lateral position, the weight of the lung and mediastinal structures cause a shift of these structures into the inferior pleural cavity. Obviously, this is the result of the weight of the structures and the effect of gravity. The same mechanical situation does not occur in the supine position and accordingly the mediastinum does not shift so readily. The supine position is better tolerated by the patient than is the lateral.

TABLE I. RESULTS IN FIFTEEN PATIENTS WITH OPEN PNEUMONOLYSIS

NAME	MONTHS AFTER OPERATION	SPUTUM	GASTRIC CONTENT	INTRAPLEURAL FLUID	OBLITERATIVE PLEURITIS	OLEOTHORAX	CONTRALATERAL LUNG DISEASE*	PNEUMOTHORAX	PROGNOSIS
H. F.	37	Negative concentrate	Not done	No	Yes	Yes	No	No, oleothorax	Good
J. S.	21	Negative culture 7 mo. after operation	Not done	No	Yes	Yes	Yes	No, oleothorax	Died
E. H.	35	?	Negative smear	Yes, culture positive	No	No	No	Yes, effective	Good
C. R.	34	Negative direct smear	Not done	No	No	No	No	Yes, effective	Good
L. L.	25	?	Negative culture and guinea pig	Yes, bilateral positive culture, operated side	Yes	Yes	Yes, but controlled	Yes, contralateral side	Good
V. S.	25	Positive smear	Not done	Empyema, contralateral side	No	No	Yes, active	No, abandoned because of contralateral lung disease	Hopeless
I. H.	23	?	Not done	No	No	No	Yes	Yes, effective	Poor
I. D.	19	Negative concentrate	Not done	No	No	No	No	Yes, effective	Good
M. M.	17	No sputum	Not done	No	No	No	No	Yes, effective	Good
G. B.	13	No sputum	Not done	No	No	No	No	Yes, effective	Good
L. L.	10	?	Positive smear	No	No	No	Yes, active	Yes, effective	Poor
J. D.	7	?	Negative smear	No	Questionable	No	No	Yes, effective	Good
H. S.	6	?	Negative smear	No	No	No	No	Yes, effective	Good
R. M.	3	Negative smear	Not done	No	No	No	No	Yes, effective	Good
W. U.	2	Not done	Not done	No	No	No	No	Yes, effective	Good

*There was no contralateral spread or activation of disease caused by operation.

The third interspace is selected for the incision. It gives the best exposure. One is sometimes tempted to use the second or fourth interspace, but if this course is followed the exposure is jeopardized. If the exposure is not adequate the adjacent costal cartilage may be divided. A division of the third costal cartilage allows more exposure in the apex, whereas division of the fourth costal cartilage permits greater exposure inferiorly. The breast is elevated superiorly. The skin, breast, fat, and pectoralis major muscle are raised as a single flap. This places the muscle incision inferior to the incision through the interspace.

The lung is always handled with great care in order to eliminate trauma. The adhesions are released in the endothoracic fascial plane, as pointed out by Eloesser[10] and Carter.[13] This step prevents injury to the lung. Much attention is given to hemostasis. All bleeding points are controlled by suture ligatures of silk. The galvanocautery or electrosurgical methods are not used because hemorrhage control is not dependable and the extent of tissue damage is greater than by sharp dissection and suture ligatures. It has never been necessary to ligate the lung side of an adhesion.

No attempt is made to close the pleura or the intercostal space. Re-expansion has not been a problem since patients with endobronchial disease which produces a check valve mechanism are not suitable candidates for pneumothorax and therefore open pneumonolysis is not needed. Bronchiolar spasms can be relieved by the use of adrenalin in oil.

In this small series, as in all other series where pulmonary tuberculosis is treated, disease in the contralateral lung reduces the chances for favorable end results. It appears that obliterative pleuritis will occur rather frequently after open pneumonolysis. Some of the patients recently subjected to operation are likely to develop this complication. It is possible that the judicious use of oleothorax will control the obliterative pleuritis.

CONCLUSIONS

1. Open pneumonolysis is an adjunct to closed pneumonolysis and pneumothorax.

2. Open pneumonolysis can be accomplished with a low morbidity and mortality rate if strict adherence to the proper indications is observed.

3. There is a definite need for open pneumonolysis in the treatment of a small group of properly selected patients with pulmonary tuberculosis.

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THE INITIAL SURGERY OF THORACOABDOMINAL WAR INJURIES

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THE term, "thoracoabdominal wound" denotes injury to the thorax and abdomen by the same missile. Such a wound necessitates actual penetration through the diaphragm or transmission of sufficient force to produce injury in the other serous cavity. The vast majority of thoracoabdominal war injuries are due to a missile actually traversing the diaphragm. Casualties with separate wounds of the abdomen and thorax present a different problem and these are better termed "combined thoracic and abdominal injuries." This discussion is limited to true thoracoabdominal wounds.

DIAGNOSIS

A working diagnosis is readily apparent in the majority of war wounds, including thoracoabdominal injuries. A thorough inspection of all surfaces of the thorax and abdomen, both anteriorly and posteriorly, for wounds of entrance or exit, plus the usual physical examination and roentgenologic procedures, is sufficient in most cases. A complete preoperative diagnosis is desirable, but the one point of utmost importance is the determining beyond reasonable doubt of the presence or absence of a thoracoabdominal wound. If such an injury is deemed to be possible from the inspection of the wound, physical examination, and a study of the roentgenograms, exploration must be done.

Thoracic wounds frequently give rise to pain and spasm of the upper abdomen and such findings are, therefore, not reliable indications of abdominal injury. Differential diagnosis in these cases by paravertebral procaine intercostal block is time consuming and often inconclusive. Thoracoabdominal wounds with the abdominal injury limited to the liver, spleen, or kidney may present a paucity of abnormal abdominal findings. Absence of peristalsis is indicative of bowel injury. However, we have noted peristalsis even in the presence of colonic injury sufficiently often to vitiate its being considered as pathognomonic. Reliance should be placed mainly on inspection of the wound, or wounds, and the projection of the suspected course of the missile from the position of the wounds, if perforating in type, or from the wound of entrance and position of the foreign body by roentgenography in the penetrating type of injury.

The pleura can be assumed, with reasonable accuracy, to follow the outline of the periphery of the ribs and costal cartilages. For tentative diagnostic purposes, the thickness of the diaphragm can be dispensed with and the peritoneum assumed to be in contact with the diaphragmatic pleural surfaces. If the diaphragm were immobile and formed a perfectly horizontal partition,

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There are occasional cases with very bizarre missile courses due to the foreign body striking a bony structure tangentially, but these are greatly in the minority. *Many such "bizarre" pathways are explained by determining the position the casualty was in when injured.*

As approximately 90 per cent of casualties with thoracoabdominal wounds show the path of the missile to be from chest to abdomen[1] and only 10 per cent from abdomen to chest, the greatest difficulty is encountered, from a diagnostic standpoint, in determining the presence or absence of abdominal injury in a patient with a known thoracic wound. As stated previously, the diagnosis is often straightforward. Exploration must be done in those in whom abdominal injury is suspected if there is any reasonable doubt. The only ones that permit a different course are those with minor liver penetrations. If such a foreign body be only 2 or 3 mm. in diameter and can be shown to lie within the liver roentgenographically, certain of these lesions may be treated expectantly. *From personal knowledge, it appears that about 1 per cent of all* thoracoabdominal war wounds fall in this category. If the foreign body is over 3 mm. in diameter it is best to determine surgically the exact damage that has been done.

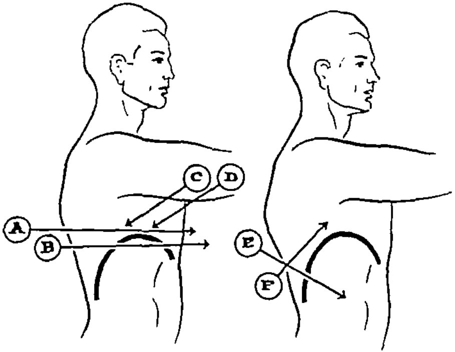

Fig. 2.—A schematic representation of the possibilities of diaphragmatic injury. Missiles entering the body at certain levels may or may not involve the diaphragm depending on the direction of the missile and the position the casualty was in when injured.

It is apparent from the foregoing that most questionable thoracoabdominal injuries that demand exploration, but where no diaphragmatic penetration is found at operation, will have thoracic without abdominal involvement. In those cases, it is possible to rule out intra-abdominal lesions by thoracic exploration and, at the same time, carry out any indicated thoracic repair. *Because of the difficulties of accurate diagnosis in lesions around the* diaphragmatic sulci and the importance of treating surgically any patient with possible abdominal injury, many exploratory thoracotomies must and should be done. In a series of 2,267 penetrating and perforating injuries of the thorax, including 903 thoracoabdominal wounds treated by the Second Auxiliary

Surgical Group, it was found[1] that thoracoabdominal injury, either suspected or proved, was the most frequent indication for thoracotomy in the forward area. In this group 122 thoracotomies were done for suspected thoracoabdominal injury where no diaphragmatic lesion was discovered at operation. Thoracotomy for suspected thoracoabdominal injury, but none found, comprised 28 per cent of all the thoracotomies done in the forward area by the group.

PREOPERATIVE PREPARATION

Resuscitative methods and proper preoperative care are of utmost importance to both the thoracic and abdominal casualty. If the wound is a thoracoabdominal one, certain concessions may have to be made that are not ideal from one or the other viewpoints. Thoracic physiology should be restored to as nearly normal as possible before operation, and the length of time this takes, within limits, is unimportant from the thoracic standpoint. Time is of great importance in cases with abdominal injury. It is, therefore, often necessary to treat the patient with a thoracoabdominal injury somewhat differently than holds for either the straight thoracic or abdominal case. Replacement therapy with blood, plasma, and electrolytes can be used quite freely for abdominal cases, but there is some danger of overloading the already deranged cardiorespiratory system of the thoracic casualty. Indicated replacement therapy should be used, and it will be found to be indicated in practically every thoracoabdominal casualty. It is necessary, though, that the shock officer and surgeon be aware of the danger of giving too much too fast. In general, one tends to operate upon the thoracoabdominal or abdominal injury as soon as it is felt that the patient will tolerate the procedure, in order to minimize the chance of peritoneal contamination. In straight thoracic cases the patient may be given a short time to stabilize after shock is, for the most part, corrected.

Two other factors need to be mentioned in regard to resuscitative measures for thoracoabdominal cases. The first is the influence of disturbed cardiorespiratory physiology on the production and prolongation of shock. The second is a similar influence due to severe peritoneal or pleural cavity contamination. The main causes of a disturbed cardiorespiratory balance in the recently injured man are as follows: the presence of blood and/or air in the pleural cavity with diminution of the functioning pulmonary parenchyma; the presence of large, pulmonary hematomas; the splinting effect of pain from an injured thoracic cage; and excessive blood or mucus in the tracheobronchial airway. All of these factors have been amply discussed previously and need not be dwelled upon here except to emphasize their importance in thoracoabdominal injuries. Their correction by thoracentesis, intercostal nerve block, and tracheobronchial aspiration is indicated as for the patient with only a thoracic wound.[2, 3]

In certain thoracoabdominal cases the patient will continue to exhibit a low blood pressure, rapid pulse, and the associated signs of shock even after correction of the thoracic physiology and adequate replacement therapy. Some of these have massive pleural or peritoneal contamination from an injured stomach or colon. In such instances, it may be necessary to proceed with surgery, as shutting off the leakage is the only way of improving their condition. It is the patients in this group who are the worst surgical risks and account for some of the operative deaths. It was always the policy to operate upon every patient, no matter how slight the hope of recovery. In only one instance in this series was operation not done because of the poor condition of the patient. This case has been reported in more detail elsewhere.[4]

Shock therapy must be continued during and after operation. It is, therefore, wise to insert a needle, or cannula, in the saphenous vein preoperatively, because it is not as likely to become dislodged by the necessary manipulations of the patient as if it were in a vein of the arm.

A Levine tube for stomach decompression is best inserted soon after admission as many patients have surprising degrees of gastric dilatation which, by elevating their diaphragm, further hampers adequate oxygenation. Also, the period of time since ingestion of food is not a good criterion of the amount of material in the stomach since a large residue is often present many hours after eating. This may be due to pylorospasm or lessened gastric motility from the nervous tension of battle.

ANESTHESIA

It is unnecessary to emphasize before this Association the importance of proper anesthesia by a skillful anesthetist. Endotracheal oxygen-ether by the closed method was our choice.[5] Periodic aspirations of material from the trachea and bronchi were essential. Positive pressure was useful to expand the lung periodically during operation and to encourage pulmonary expansion at the end of operation, especially if the thorax was closed without drainage.

APPROACH

In selecting the operative approach, prime consideration should be given to the expected intrathoracic and intra-abdominal damage. Many injuries can be repaired either abdominally or transdiaphragmatically through the thorax. Although each case must be decided upon individually, certain factors should be given consideration in arriving at that decision. There are distinct advantages to the thoracic approach for operations, that can be done from above. These can be enumerated, briefly, as follows: It facilitates repair of some upper abdominal organs, such as the spleen and upper portion of the stomach. Colostomies can be done, when indicated, through a muscle-splitting incision at a greater distance from the main incision than is possible through the abdominal exposure. Patients can be carried in a lighter plane of anesthesia, as abdominal relaxation is not necessary. The diaphragm can be more easily and satisfactorily repaired from above than from below. In patients with marked pleural contamination, the pleural cavity can be thoroughly washed out with saline solution to reduce the degree of infection. The postoperative course

Surgical Group, it was found[1] that thoracoabdominal injury, either suspected or proved, was the most frequent indication for thoracotomy in the forward area. In this group 122 thoracotomies were done for suspected thoracoabdominal injury where no diaphragmatic lesion was discovered at operation. Thoracotomy for suspected thoracoabdominal injury, but none found, comprised 28 per cent of all the thoracotomies done in the forward area by the group.

PREOPERATIVE PREPARATION

Resuscitative methods and proper preoperative care are of utmost importance to both the thoracic and abdominal casualty. If the wound is a thoracoabdominal one, certain concessions may have to be made that are not ideal from one or the other viewpoints. Thoracic physiology should be restored to as nearly normal as possible before operation, and the length of time this takes, within limits, is unimportant from the thoracic standpoint. Time is of great importance in cases with abdominal injury. It is, therefore, often necessary to treat the patient with a thoracoabdominal injury somewhat differently than holds for either the straight thoracic or abdominal case. Replacement therapy with blood, plasma, and electrolytes can be used quite freely for abdominal cases, but there is some danger of overloading the already deranged cardiorespiratory system of the thoracic casualty. Indicated replacement therapy should be used, and it will be found to be indicated in practically every thoracoabdominal casualty. It is necessary, though, that the shock officer and surgeon be aware of the danger of giving too much too fast. In general, one tends to operate upon the thoracoabdominal or abdominal injury as soon as it is felt that the patient will tolerate the procedure, in order to minimize the chance of peritoneal contamination. In straight thoracic cases the patient may be given a short time to stabilize after shock is, for the most part, corrected.

Two other factors need to be mentioned in regard to resuscitative measures for thoracoabdominal cases. The first is the influence of disturbed cardiorespiratory physiology on the production and prolongation of shock. The second is a similar influence due to severe peritoneal or pleural cavity contamination. The main causes of a disturbed cardiorespiratory balance in the recently injured man are as follows: the presence of blood and/or air in the pleural cavity with diminution of the functioning pulmonary parenchyma; the presence of large, pulmonary hematomas; the splinting effect of pain from an injured thoracic cage; and excessive blood or mucus in the tracheobronchial airway. All of these factors have been amply discussed previously and need not be dwelled upon here except to emphasize their importance in thoracoabdominal injuries. Their correction by thoracentesis, intercostal nerve block, and tracheobronchial aspiration is indicated as for the patient with only a thoracic wound.[2, 3]

In certain thoracoabdominal cases the patient will continue to exhibit a low blood pressure, rapid pulse, and the associated signs of shock even after correc-

TABLE II. SIDE OF INJURY

SIDE	CASES	DEATHS	MORTALITY IN PER CENT
	A. 903 Cases		
Right	435	103	23.5
Left	448	136	30.3
Bilateral	20	8	40.0
	B. 48 Cases		
Right	20	3	15.0
Left	26	8	30.8
Bilateral	2	0	0.0

The difference between right- and left-sided injuries in the total group (*A*) and the personal series (*B*) is apparent. In right-sided cases the liver was frequently the only abdominal organ damaged.

series was composed of 20 on the right, 26 on the left, and 2 bilateral. The mortality rates for the larger group were 23.5 per cent for the right-sided cases, 30.3 per cent for left-sided cases, and 40 per cent for the bilateral, with a 27.3 per cent over-all rate. The corresponding figures for the personal series were 15.0 per cent, 30.8 per cent, and 0.0 per cent, respectively, with a 22.9 per cent over-all rate. The effect of multiple-organ injury is better demonstrated in the larger series than in our own because of the small number of cases in the latter group. As will be seen in Table III, the mortality for those with only one abdominal organ injured was 14.4 per cent, but this rate increased to 29.2 per cent for two organs, and 58.7 per cent for three or more organs. The corresponding figures for our own series were 8.3 per cent for one organ, 37.5 per cent for two organs, and 40 per cent for three or more organs.

Table IV is included to show the mortality for the different operative approaches. However, it is not believed to be of much clinical significance, as the operative approach is influenced by the location of the wound, or wounds. Consequently, more patients with multiple abdominal-organ involvement were treated by the abdominal approach with the necessarily increased mortality.

The grouped causes of death listed in Table V are of interest. It demonstrates that more patients with thoracoabdominal injuries die of pulmonary complications than of abdominal difficulties. This seems to point out where effort can be expended most profitably to reduce the mortality. Table VI is not included in an attempt to demonstrate that thoracic surgeons are better

TABLE III. NUMBER OF ABDOMINAL ORGANS INJURED*

NUMBER OF ABDOMINAL ORGANS INJURED	CASES	MORTALITY IN PER CENT
	A. 903 Cases	
1	451	14.4
2	226	29.2
3 and over	74	58.8
	B. 48 Cases	
1	24	8.3
2	8	37.5
3 and over	16	40.0

*The mortality rate is proportional to the number of abdominal organs injured. This does not take into account the actual number of intestinal perforations. (Small intestine and colon each treated as a single organ.)

qualified to treat these cases, but because we believe it probably was due to greater attention to the pulmonary factor and resulted in the lowered mortality rate.

SUMMARY

1. Correction of cardiorespiratory inbalance is the most important phase of preoperative preparation of casualties with thoracoabdominal injuries. Thoracentesis, intercostal nerve block, and maintenance of a free airway are the three essentials of so doing.

2. The diagnosis of thoracoabdominal injury is often difficult if the wound is in the region of the diaphragmatic sulci. Exploration should be done in any case about which there is reasonable suspicion of peritoneal as well as pleural involvement.

3. The mortality rate in thoracoabdominal injuries is directly proportional to the severity of the wound. The rate increases progressively as the number of abdominal organs injured rises. Likewise, right-sided injuries do better than left-sided ones, since in the former the liver is frequently the only abdominal organ involved. In the series of 903 thoracoabdominal injuries treated by the surgical teams of the Second Auxiliary Surgical Group, the over-all mortality was 27.3 per cent. For the severely wounded thoracoabdominal casualty surviving the first postoperative day, the syndrome of "post-traumatic uremia" is his greatest hazard. In a personal series of forty-eight cases, with eleven deaths, six of the fatalities were due to this syndrome.

4. It has been demonstrated that more patients with thoracoabdominal wounds die of pulmonary complications than succumb to complications within the peritoneal cavity. This indicates that reducing the incidence of pulmonary complications in the immediate postoperative period by careful attention to securing a completely expanded lung against an intact chest wall, and the maintenance of a free airway, should result in a lowering of the mortality rate for this group of casualties.

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DISCUSSION

COLONEL JAMES H. FORSEE, Denver, Colo.—The results obtained, as reported by Dr. Betts, in thoracoabdominal injuries represent a remarkable achievement. This type of injury represents what prior to World War II had a very high mortality rating. Adequate statistical data are not available relative to the mortality rate of this type of injury prior to World War II.

In the history of the Medical Department of World War I, the mortality rate for abdominal injuries is recorded as approximately 66 per cent, and it is doubtful that thoracoabdominal injuries had any lower mortality rate. The mortality rate of 22.9 per cent, as reported in the personal series by Dr. Betts, is an achievement which we all recognize as a highly commendable one.

This paper, however, represents the work and illustrates what can be accomplished by competently trained surgeons working in forward areas. This work represents a part of the work carried out by the surgical teams of an auxiliary surgical group functioning in the most forward hospitals during a period of thirty months of overseas service. It may be of interest to this organization to know the results accomplished by this group of surgeons.

In 3,325 intra-abdominal injuries treated by members of this group, the mortality rate was approximately 25 per cent.

As stated by Dr. Betts, the mortality rate in 903 thoracoabdominal injuries was approximately 27 per cent for the entire group.

In an experience of approximately 1,500 severe intrapleural injuries, exclusive of intra-abdominal and thoracoabdominal injuries, the mortality rate was approximately 7 per cent.

It is difficult to appreciate thoroughly, without firsthand experience, the difficulties inherent in the surgical care of such severely wounded battle casualties, and likewise to appreciate the hazards under which the people work who were charged with the responsibility for the care of these patients.

This remarkably low mortality rate is a high tribute to medical education and the surgical residency system of training in this country.

By that means we were provided with a capital stock of a very large number of competently trained surgeons who, using the adjuncts of anesthesiology, chemotherapy, and resuscitation measures, were able to achieve what heretofore had never been accomplished—the extremely low mortality rate in abdominal injuries and in thoracoabdominal injuries. It also permitted, for the first time in the history of military surgery, the concentration of emphasis on the care of severely wounded or nontransportable medical casualties. This had a tremendous effect upon the morale of the fighting troops in the front lines. They knew that if they suffered severe injuries they would have first priority in their surgical treatment.

Several members of this Association actively participated in the care of these battle casualties in the forward areas.

It was our good and great fortune to have, directing the surgical efforts of these teams in this organization, Dr. E. D. Churchill, Consulting Surgeon of the North African Theater of Operations, and later of the Mediterranean Theater. Through his direction this remarkably low mortality rate was achieved.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—Dr. Betts spoke about post-traumatic uremia in these cases. I wonder how many of these cases represent misfortunes due to blood transfusion. I would like very much if he would say something about that to us.

DR. GEORGE G. FINNEY, Baltimore, Md.—I was one of the doctors on the Okinawa operation. I have been very much interested in Dr. Betts' paper, because I tried to follow the thoracoabdominal cases as well as possible during the Okinawa campaign.

Out of slightly more than 15,000 battle casualties that were taken care of in six field hospitals, because we did not have any evacuation hospitals available for the Okinawa operation, we had 95 true thoracoabdominal cases with nineteen deaths.

There were two things that made an impression on me in the care of these cases.

In the first place, if adequate anesthesia was available, and the surgeon had had reasonably good training, the mortality rate was relatively low. I think too often the important part played by anesthesia is not realized and proper credit is not given to the good anesthetist who understands his problem and thereby saves lives.

Second, the proper use of supporting therapy cannot be overemphasized. I know that early in the operation we gave too much blood many times to patients with thoracic wounds, because in the autopsies that were done on cases where there had been injury to the thorax, or a thoracoabdominal wound, nearly 90 per cent of the autopsies had very marked pulmonary edema, and in relatively few cases was there very much hemorrhage in the thorax.

I might say in reference to Dr. Graham's remark, that during the Okinawa operation we gave 40,607 pints of whole Group O blood, without a single cross-matching. As far as I know, we had only two immediate reactions that were severe enough to indicate incompatibility of the blood. We also had a small incidence of mild chills, and at times some urticaria, neither of which was prominent. We did have a certain percentage of patients who died with uremia. I am sorry I cannot give the figures; I hope they will be available. *Certainly that figure as a whole was quite low, at least so it impressed us.*

The only other thing I would like to say is that sometimes we in the Pacific felt that we were more or less stepchildren. In the Okinawa operation, even though the surgeon of the Tenth Army had sent in repeated requests, we did not have a single thoracic surgical team in the operation. As a matter of fact, we had only three other men that I could find beside myself, who had had any particular training in thoracic surgery.

That, of course, was an unfortunate thing, particularly when it is realized that there were 31,400 battle casualties among ground forces in that operation.

I am not saying that in any spirit of criticism, but it is something we ought to think about. We hope there will be no future need for it, but the proper use of specialists, when they are available, is something that merits due consideration from those in authority.

COLONEL FRANK B. BERRY, New York, N. Y.—I had the privilege of being associated with the Second Auxiliary Surgical Group, first as the Chief Surgeon of an Evacuation Hospital, and, second, as the Consulting Surgeon of an army.

In going over the record of the Seventh Army, we analyzed 55,000 battle casualties. That does not include quite all the casualties, because there were about 5,000 more, but they are the ones upon which we had accurate statistics.

Thoracoabdominal wounds comprised 0.8 per cent of that group. To show how they were distributed, in the field hospitals thoracoabdominal cases comprised 5.3 per cent of the admissions, and in the evacuation hospitals 0.4 per cent. The evacuation hospitals were located at times within one-quarter mile of the field hospitals. At other times they might be five to ten or, in the later days of the campaign, when the final pursuit was on, twenty-five or thirty miles to the rear.

Our total mortality for this group of patients was 21.7 per cent: the field hospital mortality was 26.5 per cent, and the evacuation hospital mortality, naturally lower, 15.2 per cent.

In regard to the mismatchings on the blood transfusions that Dr. Graham spoke of, if Dr. Churchill is here, he can tell more about the Mediterranean Theater than I can. Our resuscitation and blood transfusion therapy followed the directives of Dr. Churchill. It was probably, without question, the best system of blood collection and transfusion that was planned for the entire United States Army. In the initial stages, and as long as blood was supplied within the theater, we had only low titer "O" blood, which is an important distinction from just "O" type of blood, which includes also the high titer.

With the rare exception of times when patients were in such severe degree of shock that they needed blood immediately, cross-matching was done for all patients, both in the field and in the evacuation hospitals. Cross-matching in the field hospitals continued with each succeeding transfusion after the initial transfusion.

I know that the blood, before it was taken, was all grouped and again tested from the bottles before they were sent out. In all cases, therefore, there was a double check and in some cases a triple check before the blood was given.

The only trouble we had with the blood was with *a small amount furnished us from a* collecting station near Paris, and some of the Red Cross blood coming to us from the States;

which had not been refrigerated at first and which came to us in large amounts. This was also given in some instances without proper cross-matching.

I agree with the work of Van Slyke, Richards, Homer Smith, and Evans that the kidney is one of the more sensitive organs and responds very quickly to anoxia brought about by shock, and that a great majority of the cases of anuria were due rather to the injury and a very small percentage to transfusions.

DR. MARK H. WILLIAMS, Binghamton, N. Y.—Dr. Betts has made a valuable contribution to this subject. I would like to say a few words about an experience with another Surgical Group in a different Army in the same theater.

(Slide.) Here are thirty-three thoracoabdominal wounds. It will be noted that the right-sided cases outnumber the left by about two to one. In one there was a tangential wound; the missile entered the abdomen on the left, crossed to the right, and penetrated the right leaf of the diaphragm.

The operative approach is about equally devided between laparotomy and thoracotomy. Early we used a thoracotomy approach by preference for right-sided cases and later came to prefer the same approach for the left-sided cases also.

The mortality rate is low, largely due to the preponderance of right-sided cases and also because among these we had several which were comparatively minor injuries. You will note that better than one-half of the total mortality occurred in the left-sided cases which comprise approximately one-third of the entire group.

One word about anuria. We had our largest number of cases of anuria during a period of cold weather. Of course it may have been a coincidence but many of us in the Third Auxiliary Surgical Group gained the impression that prolonged exposure to cold and delay in instituting blood replacement was an important factor.

DR. EMILE HOLMAN, San Francisco, Calif.—One episode may give us some information about the anuria. A patient who died thirty-six hours after operation and had adequate studies made, showed a hemoglobinuria in the few ounces of urine passed after the operation, and at autopsy the kidney tubules were found to be filled with hemoglobin casts. It was an obstructive phenomenon in the kidney rather than injury to the kidney itself. The hemoglobinuria and the hemoglobin casts were considered to be due to hemolysis of the transfused blood.

DR. ROBERT R. SHAW, Dallas, Texas.—Just a word about the late complications of these wounds. In working in a chest center in Paris, where I had the opportunity to see these patients roughly ten days to twenty days following the initial wounding and initial surgery, I saw forty-four patients who had complications following surgery for thoracoabdominal wounds. These complications were uniformly light suppurative.

As Dr. Betts has indicated, the mortality of the wound on the left side is higher than that on the right. However, the complications are much more prevalent on the right than on the left.

In the group of forty-four cases there were thirty-eight right-sided cases and only six left-sided cases. The higher mortality was due primarily to faulty handling of drainage from the damaged liver, which is a point that I think should be brought out. Where no drainage was instituted, or where only a very small stab wound was used subcostally to drain the damaged liver, these patients almost invariably excreted bile beneath the diaphragm which would break down the diaphragmatic repair and cause bile pleuritis. This, although it was not a great mortality factor (there were only two deaths in this group of forty-four) did constitute a large morbidity factor because it was sometimes very difficult to clear up this bile pleuritis. It almost invariably necessitated an open thoracotomy with decortication and another repair of the diaphragm with a better drainage of the damaged liver from below.

The left-sided complications were sometimes rather bizarre. In one case a soldier with an unrecognized congenital diaphragmatic hernia was wounded through the chest, so that the colon was perforated and he developed a very neat colostomy. Three days later feces were pouring out through a broken-down thoracotomy wound. His case was a complicated one that took considerable surgery, before he was finally restored to normal.

Two cases had gastropleural fistulas, rather wide-open fistula, in which the repair of the stomach had broken down and also broken down the diaphragmatic repair. These were difficult, because the skin became badly excoriated from the irritating gastric secretions.

The mortality rate of the complications was low. However, they were a rather significant group in the patients seen in the chest centers behind the lines.

DR. A. L. LOCKWOOD, East Toronto, Ontario.—May I ask Dr. Betts just two questions? Could he give us any idea of the percentage of these wounds that were shrapnel rifle bullet or machine gun, and whether or not the use of sulfonamide drugs had anything to do with uremia?

DR. EDWARD D. CHURCHILL, Boston, Mass.—I wish to comment on the deaths in battle casualties accompanied or caused by renal failure. Dr. Graham has asked a very pertinent question, which we asked ourselves many, many times. Just how often was blood transfusion responsible?

The story of transfusion in an overseas theater is a simple one. We found ourselves in North Africa fighting the war with plasma, and no facilities beyond Erlenmeyer flasks and sodium citrate for transfusion.

The surgeons working in the forward area were up against it. Their personal diaries, some of which I have had the privilege of reviewing recently, repeat and repeat the desperate need of whole blood. Stories are recorded in these diaries of the surgeons themselves, after operating upon a patient, giving their own blood to pull their patients through. Now, that obviously could not go on. We had to have blood. This was in 1943. At that time we were isolated in the center of the Mediterranean. There was no possibility of obtaining whole blood from the United States, so we proceeded to plan our own blood bank system.

We did that in two phases, as it was necessary to wait for refrigeration equipment to be shipped. It sounds simple, but it takes at least six months to get supplies from the United States into distribution in an overseas theater.

For a while the evacuation hospitals organized their own banks, securing donors from nearby service organizations or the lightly wounded. That left the field hospitals, with surgical teams, out of the picture as these light mobile units had neither the personnel nor the equipment to run blood banks. Also, their only source of donors could be combat troops. Yet they needed blood most desperately for the seriously wounded, including such cases as thoracoabdominal injuries.

When supplies were received it was possible to organize a large blood bank and a distributing system that supplied the field hospitals completely, and, in addition, materially lightened the load of the evacuation hospitals.

In early February, 1944, the blood bank in Naples started its work. More elaborate safeguards were imposed on the processing of the blood than are customary in civilian hospitals. A seven-day time limit was set on preserved blood. Sodium citrate and glucose were used as the preservative and special precautions were taken to keep the blood at the proper reduced temperature from the time of drawing to the moment of use. Under these conditions the blood was as perfectly preserved as with the ACD solution later proposed by the National Research Council.

"O" blood was tested for its isoagglutinin titer and that with titer above 1:64 was used only for "O"-type recipients. Universal donor blood was therefore "O" type with low isoagglutinin titer.

Cross-matching was demanded as a further safeguard. I trust that you will take the remark of Dr. Finney as an indication of the urgency of the situation he was facing, because I am sure that he would be the last one to recommend that a large number of transfusions, or even a single transfusion, should be given without cross-matching. That we insisted upon.

To interpret the case of a patient with anuria, blood given in transfusion must be, like Caesar's wife, completely above suspicion. It must be compatible both in type and isoagglutinin titer. It must be preserved by constant refrigeration. It must be collected and stored in a closed system to avoid contamination. In military operations frequent checks must be made on free hemoglobin content both at the bank, in the forward hospital, and by examination of recipient's plasma after transfusion.

It is our belief that we have observed a sufficient number of wounded with anuria to establish the condition as an entity not caused by transfusion.

It is premature to speculate on the etiology of the anuria of the seriously wounded or assign this or that factor as the cause. It appears wise at this moment to refer to it as pigment nephropathy. Whether it is obstructive, as Dr. Holman says, is questionable. Probably there is more to it than simple obstruction of the tubules by pigment. Whether it is caused by the anoxia to which the kidney is exposed during a prolonged period of shock, as Dr. Berry suggested, based on the work of Van Slyke, is again open to question. The diffuse cortical necrosis of the kidney following experimental occlusion of a renal artery appears to differ microscopically from the condition under discussion.

Material and data have been collected in the combat area and are still under study. Opinions should be reserved until facts based on observational data are available.

In the meantime, I am perfectly aware, and I am sure Dr. Graham is aware, that misleading case histories are being reported in the literature. All one has to do is to read the evidence and say, ''In this case they have not excluded the possibility of incompatible blood transfusion or transfusion with poorly preserved blood.'' There are many of these cases already recorded in the literature.

It does seem quite clear that Bywater's work on myoglobulin pigment, in so far as it pertains to the crush syndrome, is probably correct. In the other group, the men with extensive wounds without crush, we are not yet prepared to say that the pigment is entirely myoglobulin, or whether focal injury in the kidney resulting from shock is responsible for the pigment localizing in that particular portion of the tubule.

More experimental study will probably be necessary before a solution is found.

DR. REEVE H. BETTS, Boston, Mass.—I am in complete agreement with practically everything that has been said, and many of the points would have been mentioned originally if time had permitted. I will answer only one or two specific questions.

Dr. Churchill has covered more adequately than I can the question raised by Dr. Graham concerning the post-traumatic uremias. We were fortunate in the Mediterranean theater in having most of our pathology under the direction of Colonel Mallory, who made some careful observations in the field on these patients both before and after their fatal termination.

Regarding Dr. Lockwood's question, I cannot give the exact figures, but shrapnel or high-explosive shell fragments accounted for approximately 75 per cent of the injuries and bullets for only about 20 to 25 per cent.

Sulfanilamide was used routinely in the treatment of all wounds in the forward areas early in the campaign. Late, after penicillin became available, the use of sulfanilamide was at the discretion of the surgeon.

In regard to giving transfusions without cross-matching, Dr. Berry has already indicated our practice in this regard. If the patient was in severe shock and we believed added risk was entailed by waiting for a cross-matching before transfusion, one or sometimes two transfusions were given while the laboratory was typing and cross-matching. In most instances, by the time 500 or 1,000 c.c. had been administered, cross-matched blood was available for any further needs of the patient.

ARTERIAL INJURIES OF THE THORAX

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IN THE care of large numbers of major casualties among marine and naval personnel from the Gilberts, the Marshalls, the Marianas, Iwo Jima and Okinawa campaigns, many complicated and interesting types of injuries were encountered. Among the most serious injuries were those of the major arteries, the so-called false aneurysms and arteriovenous aneurysms, or a combination of both. Technically the surgical management of some of the more proximal arterial injuries was undoubtedly one of the most hazardous and difficult operations in war surgery. Those in the extremities distal enough so that a tourniquet could be used and a direct dissection done in a bloodless field were relatively simple. Those injuries located in the groin, the abdomen, the neck, shoulder, and chest, however, required an entirely different approach and surgical management if the patients were to survive their operations. This was particularly true of the arterial injuries of the thorax, with its close anatomic relationships and difficulty of obtaining wide and easy exposure.

Wounds of the thorax, whether due to fragments, bullets, bayonets, or knives, either major or minor, have the serious potentiality of an injury to one of the many arteries, both superficial and deeply located in the thorax. It is difficult to estimate the incidence of this type of injury since most of these wounds were undoubtedly immediately fatal. This is due to the fact that a tourniquet could not be used and, likewise, adequate pressure control was rarely instantly available or possible. This type of injury, however, was certainly not always fatal because some of these patients reached base hospitals at considerable distances for definitive treatment. The hemorrhage was at least temporarily controlled by infiltration of the surrounding muscles and tissues, with a resulting pulsating hematoma or false aneurysm.

The diagnosis was frequently missed because of the insignificance of the external wound and the lack of obvious extensive infiltration or hematoma, or visible or palpable pulsation. Many were not diagnosed until an episode of serious secondary hemorrhage occurred. It became absolutely essential that every wound in this region be examined specifically for evidence of arterial injury, especially by auscultation for the presence of a bruit. Because of the serious nature of the operation necessary to cure this type of injury, surgery should be withheld long enough to insure a reasonably certain diagnosis, ruling out bruits resulting from extrinsic infiltration and pressure on the artery, and also functional bruits. Operation should not be delayed long, however, in order to avoid serious secondary hemorrhages and pressure atrophy of nerves and muscles, with the associated great loss of function.

From the Department of Surgery, the Lahey Clinic.

Read (by title) at the Twenty-sixth Annual Meeting of The American Association for Thoracic Surgery, Detroit, Mich., May 29, 30, and 31, 1946.

MANAGEMENT AND TREATMENT OF SPECIFIC ARTERIES

The Mammary and Intercostal Arteries.—Injuries of the mammary and intercostal arteries were of two general types, (1) false aneurysms with *subpleural* hematoma and infiltration, with delayed external secondary hemorrhage, and (2) those associated with a pleural laceration and continued *intrapleural* hemorrhage. This latter injury produced symptoms and signs of an increasing intrapleural pressure and mediastinal shift which progressed until death ensued in spite of oxygen and thoracentesis unless the chest wall artery was ligated. This is in direct contrast to hemothorax due to lung injury which was usually controlled by the pulmonary tamponade from the associated hemothorax. Massive external secondary hemorrhage and signs of uncontrollable, progressive, intrapleural hemothorax demand immediate exploration of the wound and careful ligation of the mammary and intercostal arteries involved. This could not be done satisfactorily or effectively except by partial removal of the costal cartilages or ribs in this region. The operation is relatively simple and no patient should be allowed to die from this type of injury.

The Axillary Artery.—Since the axillary artery is superficial, there was usually a pulsating mass and the bruit was quite obvious both by palpation and auscultation. These wounds were usually associated with signs of nerve injury due either to direct injury of the axillary cords by the same missile as that which produced the arterial injury or to the associated infiltration and pressure of the hematoma. Even though the external wound was frequently very small, the danger of secondary hemorrhage was great in this type of wound and these patients should be operated upon at once. In order to do this type of operation safely, it is essential to have a fully equipped operating room, a trained anesthetist, adequate assistants, and continuous transfusions throughout the procedure. It was found best to start the operation by tying cannulas in the ankle veins of both legs so that if one transfusion should plug up at a crucial time during the operation, the other would carry on satisfactorily, or if a very severe hemorrhage was encountered, both could be run in as rapidly as possible.

The general principle of approach to this type of injury was to avoid entering the false aneurysm at any point before the major artery and vein, both proximal and distal to the injury, and all significant branches entering into this section of the injured vessel were fully controlled. This was accomplished by a complete and accurate exposure of these vessels proximal and distal to the infiltrated area of the false aneurysm and control of the flow through these vessels by tension on small soft catheters placed around these vessels. The vessels were then further exposed until finally the false aneurysmal sac was entered and the actual injury to the artery visualized. Even with full control of the injured major artery and vein both proximal and distal to this area and all major entering branches to this area, there was still a very rapid flow of blood from the injured area into the sac. The artery and vein were then ligated carefully, both distal and proximal to the injury, and the injured area was excised, carefully preserving for collateral circulation all branches which did not immediately enter into the area to be excised.

For the axillary injuries, the approach was through an incision from the middle third of the clavicle across the pectoral region to the axilla (Fig. 1). Usually the pectoral muscle was so infiltrated with hematoma and so indurated that simply splitting the fibers would not have given adequate exposure of the vessels proximal to the injury without inadvertently opening the false aneurysm, which would immediately prove fatal. Adequate exposure could be obtained only by sectioning the pectoralis major in its tendinous insertion on the humerus, and turning it back, giving a full exposure of the axillary contents. The artery and vein were then isolated carefully just below the clavicle and catheters passed around these structures for tourniquet action. These vessels were then likewise isolated distal to the false aneurysm, usually at the margin of the axilla, and catheters passed around the vessels at this point. The pectoralis minor was then sectioned and turned downward, giving a complete exposure of the axillary vessels. A gradual dissection and development of the vessels was then carried

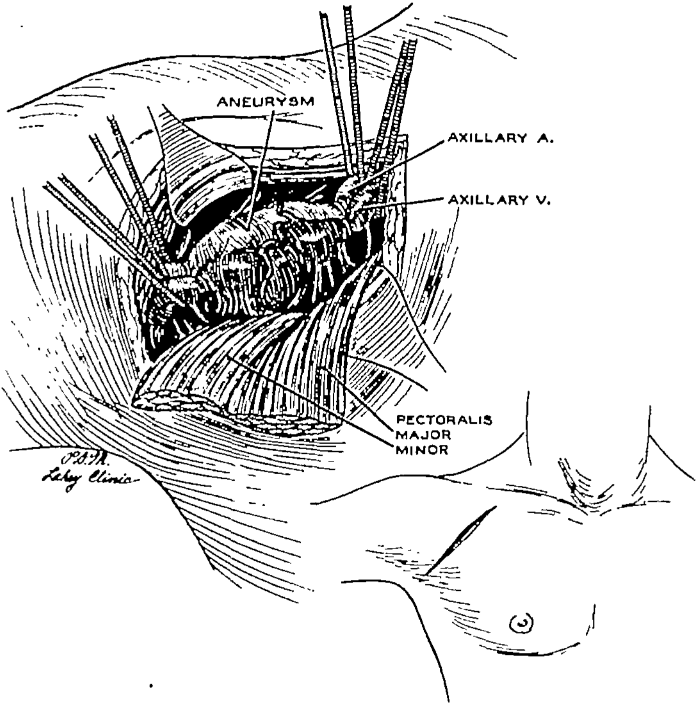

Fig. 1.—Exposure of the axillary aneurysm, and approach and control of the axillary vessels; insert shows line of incision.

out both from above and below, carefully isolating and preserving the axillary cords which are in close contact with the vessels. Finally, the neck of the false aneurysmal sac was encountered and it was then necessary to open the sac and accept a certain amount of rather brisk hemorrhage while the remainder of the artery was isolated and the ligation carried out both distal and proximal to the laceration, and the injured area excised.

In many instances the vein was involved and there was an arteriovenous aneurysm in addition to the false arterial aneurysm. In those cases it was absolutely essential that the vein be excised as well, but this is equally important in those cases in which the vein is not involved, since this procedure facilitates the circulatory adjustment following the ligation of the artery. No attempt was made at this operation to suture lacerated or injured axillary nerve cords. The pectoralis major muscle tendon was then reunited with silk sutures and the wound closed, draining the wound with a small Penrose drain.

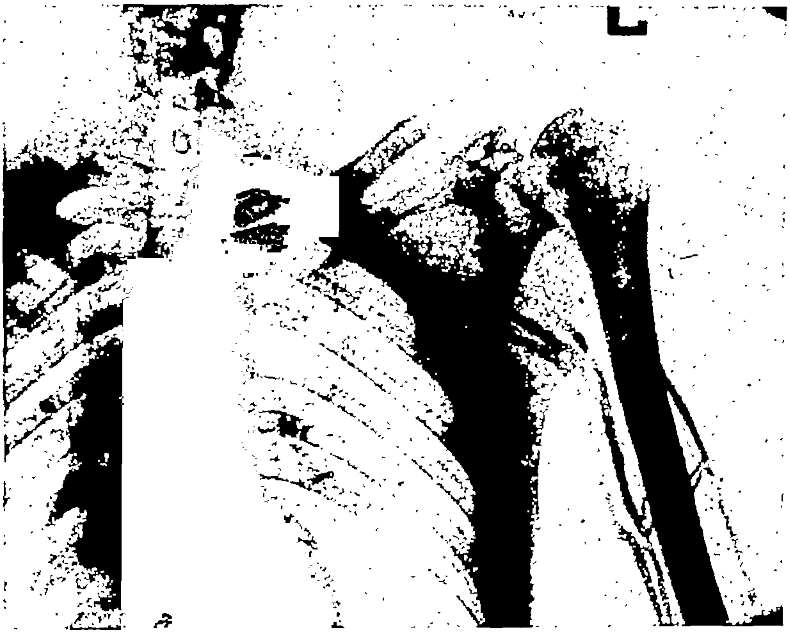

Fig. 2.—False aneurysm in conjunction with arteriovenous aneurysm of the subclavian vessels; venogram showing block of the vein at the site of injury.

The Subclavian Artery.—Injury to the subclavian artery was quite similar in many ways and in diagnostic findings to the injuries of the axillary artery. Being more deeply seated, the external infiltration and hematoma were much less obvious and the diagnosis was made chiefly by the presence of a bruit. This was a rough bruit throughout systole or during the entire cardiac cycle if combined with an arteriovenous aneurysm. It was a common finding in both the axillary and subclavian arteries to have not only a false aneurysm but an arteriovenous aneurysm as well. This added complication of the arterial injury could often be diagnosed by an increase in the venous pressure in the arm involved, and the venograms were most characteristic (Fig. 2).

Functional bruits are fairly common in this region and must be distinguished carefully. This can be done by examining the patient frequently over a period of time. A functional bruit will vary considerably from examination to examination and also in relation to the position of the patient and the position of the extremity. Likewise, simple compression from external pressure and infiltration about the artery may produce a bruit. It is, again, essential to distinguish, if possible, between compression and the true lacerated vessel with a false sac, in which surgery is essential to effect a cure. If there was any question regarding the diagnosis of the type of bruit heard, that is, whether it was due to a false aneurysm or to a combination with an arteriovenous aneurysm, or whether it was due to external pressure or a functional bruit, it was usually safe to keep these patients in bed under observation, and as long as there were no signs or symptoms of further infiltration or cardiac damage, a careful period of observation could be carried out. The functional bruits could be ruled out by their inconstant nature; the bruits due to pressure gradually decreased in intensity as the external signs of infiltration diminished and the patient improved.

Although the false aneurysms and arteriovenous aneurysms of the subclavian artery were most hazardous surgical problems, the same general principles of approach applied as described under the procedure for axillary arterial injuries. It was essential first to make a low cervical incision above the medial end of the clavicle (Fig. 3). The lateral clavicular insertions of the sternomastoid muscle were cut, the carotid sheath and its contents retracted medially, the scalenus anticus muscle was exposed, and the phrenic nerve freed and retracted medially. The scalenus muscle was then cut, the subclavian artery exposed, and a catheter passed around it for tourniquet action. Connecting this incision, a diagonal incision was then made across the pectoral region similar to that for an axillary exposure except that the pectoral muscles were split for exposure of the axillary vessels immediately below the clavicle and catheters passed around them for control. The clavicle was then cut across and the ends retracted, giving a wide exposure of the injured area. Dissection of the subclavian vessels was then carefully carried out in both directions until the neck of the sac of the false aneurysm was isolated and entered, here again accepting considerable bleeding in the final identification of the actual opening in the artery. The artery and vein were then ligated proximal and distal to this laceration and the injured area excised.

The brachial cords were, of course, closely associated with the aneurysmal sac and the missile had often done direct damage to these nerves and further damage could be demonstrated from direct pressure and the infiltration of these trunks. It was necessary to isolate them carefully and produce no operative trauma to these cords, but no attempt was made to suture any of the cords that were found injured. The clavicle was then wired together and the wound closed, draining the wound with a simple Penrose drain.

The Innominate Artery.—The diagnosis was made primarily on the basis of a bruit and roentgenologic findings of infiltration of the superior mediasthe entire superior mediastinum and the arch of the aorta. The innominate artery was isolated at its origin from the arch of the aorta and a catheter passed around it for tourniquet action. The innominate vein also was isolated and controlled in a similar way. Just beyond the bifurcation of the innominate vessels, the subclavian and the common carotid arteries and their associated veins were isolated and likewise controlled with catheters. Finally, with full control both proximally and distally by means of traction on the catheters, the aneurysmal sac was isolated and the injured section of the artery excised after careful ligation both proximal and distal to the laceration. The manubrium was then wired together and the superior mediastinum drained with a simple Penrose drain.

SUMMARY AND RESULTS

There were fourteen cases of false aneurysm, many in combination with arteriovenous communications as well—six axillary, six subclavian, and two innominate. There were no deaths or complications aside from occasional swelling and weakness of the upper extremity. No amputations were necessary and no treatment was required for any unusual circulatory disturbance in this respect. The patients recovered rapidly. Return of function of the upper extremity was variable, depending on the degree of the original associated nerve and muscle damage. The lack of circulatory complications was undoubtedly the result of the age of these patients and most probably this procedure could not be done in an older group of patients without more serious circulatory complications.

Erratum

On page 290 of the August, 1946, issue, the article "Esophagotracheal Fistula Due to Nonpenetrating Crushing Injury" by Commander Herbert D. Adams and Commander Roy E. Mabley, Medical Corps, U.S.N.R., the name of the second author should be spelled Mabrey.

JUST ISSUED

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By Andrew Ladislaus Banyai, M.D., F.A.C.P., F.C.C.P.

Associate Clinical Professor of Medicine, Marquette University Medical School, Milwaukee, Wis.; Member, Editorial Board, "Diseases of the Chest"; Formerly Preceptor in Tuberculosis, School of Medicine, University of Wisconsin, Madison, Wis.

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Pneumoperitoneum is a technically simple and safe procedure. Every physician with average manual dexterity should be able to give this treatment. As a matter of fact, it has been proposed, and I believe justly so, that pneumoperitoneum treatment of ambulatory patients should be carried out by general practitioners whenever given circumstances so require. *I maintain that reading over carefully the outlines of its technique should enable one to give pneumoperitoneum without any difficulty.*

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"All Christmas Seal Stamps have been turned into cash!"
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By giving the greatest gift of all.*
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Fifties and hundreds and thousands a-plenty.
And then, in a twinkling, it all became clear
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—There'd be funds for research and the quick diagnosis
To prevent and control tuberculosis.
—Case-finding clinics for constant inspections
To cut down the rate and the root of infections.
—Vocational counselling to guide us back
And keep us on the healthy track.
So we closed our eyes with a grateful smile,
And slept and dreamed for a little while.
And God, whose job it is to nurse
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Thoughtfully nodded His kindly head
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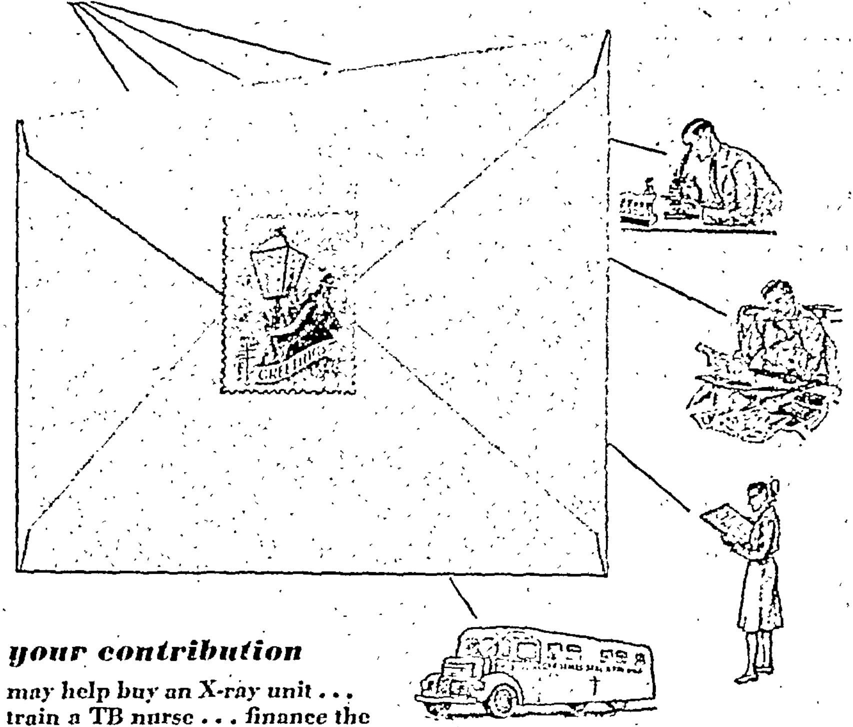

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The Journal of Thoracic Surgery

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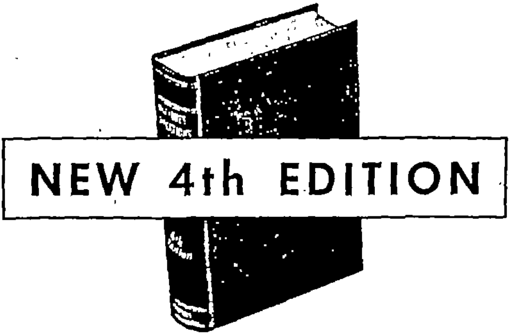

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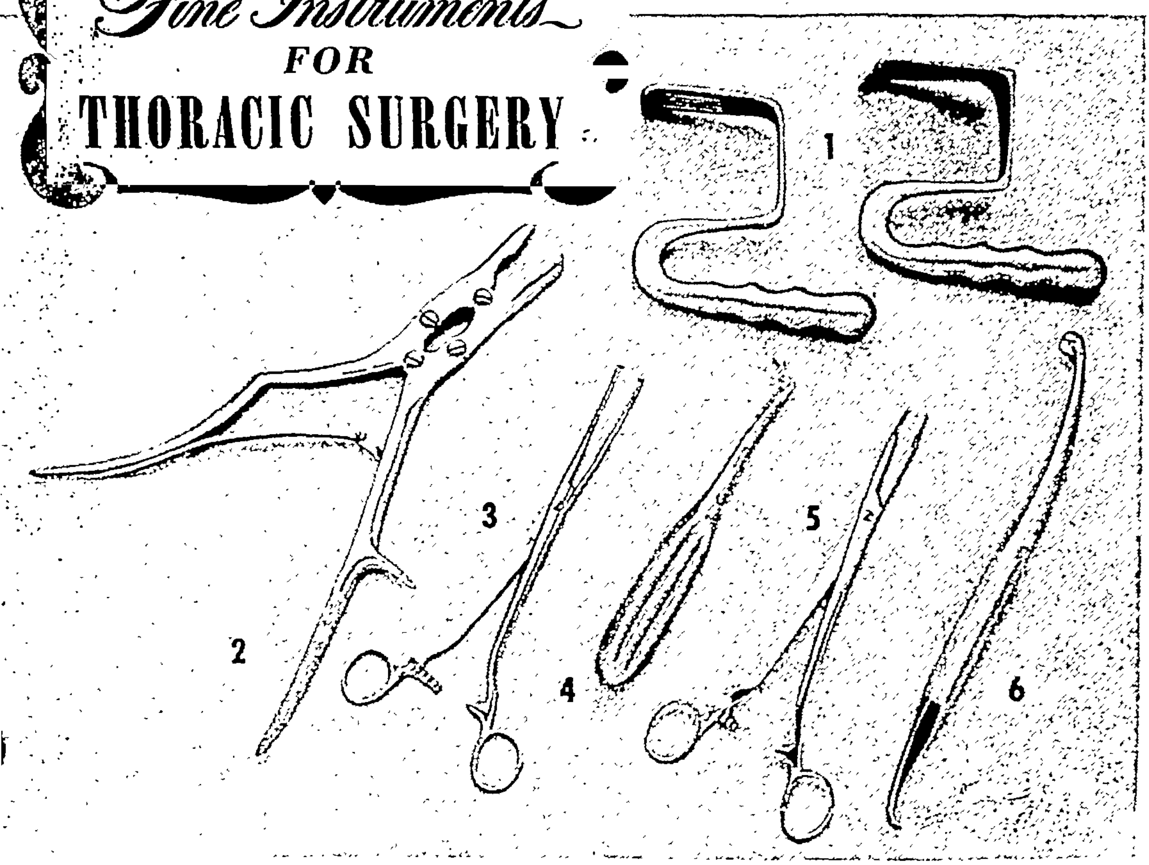

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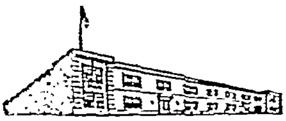

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Vol. 15 June, 1946 No. 3

Original Communications

INTRACAVITARY (MONALDI) SUCTION

Jerome R. Head, M.D.
Chicago, Ill.

INTRACAVITARY suction (Monaldi) has been employed at the Edward Sanatorium for the past three years. During this time it has come to occupy a definite and important place in the treatment of pulmonary tuberculosis. The early hope that it would prove an easy way of closing cavities and would therefore supplant some of the older forms of therapy has not been fulfilled. The difficulty in establishing it in small cavities, its uncertainty, and the long period which it must be continued have limited its use to those patients who, because of low vital capacity, cannot be treated by pneumothorax, thoracoplasty, or extrapleural collapse. In these rather common cases in which no other form of treatment is possible it offers a possible means of arresting the disease. It is also used occasionally to reduce the size of extremely large cavities so the thoracoplasty will be more likely to close them.

Because the method is relatively new and is not widely used it seems advisable to describe the technique in detail and to present a brief summary of each case in which it has been used.

THE TECHNIQUE

When the procedure was first used it was assumed that if no free pleural space could be found with the pneumothorax needle at the site chosen for drainage, adhesions were present and the trocar could be inserted safely. In two of the early cases this assumption proved to be incorrect and the catheter was inserted through a free pleura. A fatal empyema with bronchial fistula resulted. Since that time the operation has been performed in two or three stages.

Read at the Twenty-fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

Editor's Note: Apologies are due to Dr. Head and the various discussers for the long *delay in publishing this paper and the discussions.* The paper was part of a symposium of which the leading paper was by Dr. H. McLeod Riggins on "Studies of the Pathogenesis, Dynamics and Closure of Tension Cavities." Repeated efforts have been made in vain to obtain Dr. Riggins' manuscript. Finally, it has been decided to publish Dr. Head's paper with the discussions of the symposium. Dr. Arthur Vineberg's presentation was a motion picture without a manuscript.

At the first stage the pleura is explored through a small intercostal incision, if the catheter is to be inserted from in front, or by resection of a small piece of rib, if the catheter is to be inserted from the back. If adhesions are found the wound is sutured and the catheter inserted one or two weeks later. A small amount of lipiodol or a small metal clip is left at the level of the pleura so that on subsequent x-rays the relation of the proposed site of entrance to the cavity can be determined. If a free pleura is encountered the pleura is opened and a small iodoform gauze pack is inserted just to the level of the pleura. A metal clip is attached to this for the purpose of location by x-ray and the wound is sutured. Ten days or two weeks later the pack is removed and the presence of adhesions verified. The wound is again sutured and one week later the catheter is inserted. Until fairly recently the catheter was inserted at the time of removal of the pack and was brought out through a small stab wound just below the incision. So commonly this resulted in infection and opening of the incision that the third stage was adopted.

The cavity is located first on both anteroposterior and lateral x-rays. Most apical cavities are best approached from in front through the first interspace. Small ones located posteriorly can be entered most easily from behind. Cavities located elsewhere than in the apex are approached directly at the point where they are nearest to the chest wall.

Placing of the catheter requires extreme care. The cavity is located first by exploratory aspiration with a long 19 gauge needle. This is attached to a 20 c.c. syringe. The point of the needle can be assumed to be in the cavity when air or pus can be freely aspirated. The depth of the cavity is then noted and marked on the cannula which is the type used in draining the gall bladder and of a size to admit a No. 14 French catheter. The trocar and cannula are then inserted in the determined direction to the determined depth and the catheter put in place. This is at first put in farther than is necessary and then, when the cannula has been withdrawn, is pulled out until a mark, so placed on it that the tip will be about one inch within the cavity, appears at the skin.

It is extremely important that the tube be fixed firmly in place by an air-tight adhesive dressing and that great care be taken that it is not pulled out, but that the mark on the catheter be kept always at the level of the skin.

Continuous suction is maintained through a bottle trap by a small electric pump (Stedman). This is kept up for from six to ten months. The catheter is then left in place and open for another month. If the cavity reopens, suction is again applied. If it remains closed, the tube is removed.

RESULTS

During the past three years 29 patients have been treated by this method. Of these, 16 are still living and 13 are dead. For the most part the procedure was used as a last resort in desperate cases in which the patient had too extensive involvement and too little vital capacity to permit of any other form of collapse therapy—a group in which a fatal outcome seemed almost certain and any recoveries could be classed as pure gain.

The causes of death in the 13 patients who died were as listed in Table I. The extent of cavity closure in those who died was as shown in Table II.

TABLE I. CAUSES OF DEATH

CAUSES	NUMBER
Empyema with bronchial fistula	2
Hemorrhage from the treated cavity with extensive spill	1
Tuberculous meningitis	1
Normal progression of extensive soft tuberculosis	5
Tuberculous enteritis and laryngitis	3
Cardiac decompensation	1

TABLE II. EXTENT OF CAVITY CLOSURE

EXTENT	NUMBER
Closed	3
Greatly decreased in size	7
Unaffected	3

TABLE III. RESULTS IN LIVING PATIENTS

RESULT	NUMBER
Cavity closed; sputum negative	7
Treated cavity closed; patient greatly improved	6
Cavity reopened after thoracoplasty	1
Cavity decreased in size, hemorrhage, worse	1
Unimproved with poor prognosis	1

In the 16 patients who are alive the results are as shown in Table III.

Of the 29 patients, 22 had too extensive disease and too little vital capacity to permit of any other form of treatment. Without direct treatment of the cavity the prognosis was hopeless.

The results in these 22 cases are as shown in Table IV.

TABLE IV. RESULTS IN TWENTY-TWO PATIENTS WITH EXTENSIVE DISEASE

RESULTS	NUMBER
Well	5
Greatly improved	6
Greatly improved; died of cardiac failure	1
Died of progression of tuberculosis in other lung areas	4
Died of enteritis and laryngitis	2
Died as a result of treatment	2
Alive; worse	1
Alive; unimproved	1
Total	22

In 7 of the early cases the procedure was used in patients who might have been treated otherwise. The results in these are as shown in Table V.

At first there seemed reason to hope that in patients with extensive soft tuberculosis, closure of the cavities would be followed by healing of the un-

TABLE V. RESULTS IN SEVEN EARLY CASES

RESULTS	NUMBER
Well	2
Cavity reopened after thoracoplasty	1
Died as result of treatment	2
Died of progression of tuberculosis	1
Died of tuberculous enteritis	1

excavated lesions. This hope has not been fulfilled. In nearly every instance the cavity closed or became smaller but the other lesions progressed, and after stopping the suction the cavities reopened and enlarged.

CONCLUSIONS

From the foregoing recounted experience the following conclusions are suggested:

1. Intracavitary suction should never be used when any other form of treatment is applicable.

2. It is indicated for quiescent cavities of relatively long standing which are not surrounded by soft active disease in patients in whom low vital capacity precludes the use of other measures.

3. It is of value in reducing the size of extremely large excavations as a preliminary to thoracoplasty.

DISCUSSION ON "STUDIES OF THE PATHOGENESIS, DYNAMICS AND CLOSURE OF TENSION CAVITIES" BY DR. MCLEOD RIGGINS; "THE DETERMINATION AND TREATMENT OF PRESSURE CAVITIES IN PULMONARY TUBERCULOSIS" BY DR. ARTHUR VINEBERG; AND "INTRACAVITARY (MONALDI) SUCTION"

BY DR. JEROME R. HEAD

DR. JOHN ALEXANDER, Ann Arbor, Mich.—Dr. Riggins and Dr. Gearhart have added important and much-needed fundamental information about the still somewhat obscure matter of tension cavities. The application of this information to the therapeutic management of our patients is especially difficult because of the many variable factors that are involved. For example, the dramatically successful treatment reported by Dr. Riggins, in which a cavity closed immediately upon the aspiration of air, and has remained closed for a year, is unlikely to be duplicated often in view of the fact that prolonged aspiration through Monaldi drainage tubes so rarely completely and permanently closes a cavity.

It seems likely that the effectiveness of collapse therapy in closing tension cavities depends largely upon the degree to which the strictured bronchus resists the expelling of air and secretions from the cavity during expiration or coughing, or during the collapsing effect of whatever operation is used. A "positive pressure threshold" of 15 or 20 cm. of water may become changed by collapsing operations to a higher or a lower value as a result of a change in the position of, and tension upon, the stenotic bronchus that is responsible for tension within the cavity. If this assumption is true, a preoperative determination of the positive pressure threshold in a given case would not necessarily be a constant measure of the resistance of the stricture to the discharge of the air or fluid contents from a cavity following pulmonary collapse and, therefore, would not necessarily be a measure of the likelihood of complete cavity closure.

These remarks are meant to be preliminary to what I expect to say this afternoon in the hope of combating what I believe is present overenthusiasm about pulmonary resection for tuberculosis. If thoracoplasty works as well as many of us think it does, I believe we cannot afford to substitute a high operative risk and a high percentage of spread for the remarkably good results that thoracoplasty gives in a very high percentage of cases.

DR. E. J. O'BRIEN, Detroit, Mich.—I expected to say exactly what Dr. Alexander has said, and I think some of it is worth repeating. Technically, this is a beautiful piece of work, but I do not know just how much it means, nor how much benefit either the patient or the operators obtain by needling a lot of [illegible] preoperatively. We all admit, and know, and the essayists them[illegible]ed that [illegible]y be blocked today and not be blocked tomorrow. It may c[illegible] by itse[illegible] the course of collapse therapy measures.

We have all seen a number of patients with apparently blocked cavities getting larger under artificial pneumothorax, which close when the pneumothorax is abandoned. This is especially true if a phrenic is added. The change in position apparently relieves bronchial angulation. We have also seen definite tension cavities at the apex which close readily after thoracoplasty. In fact, free drainage and normal pressures may occur after the first stage thoracoplasty. I object very much to the hazardous needling of a cavity to see what the pressure is, therefore, until the collapse measures, which we have been using successfully so many years, have been tried. In the abstract of this paper the statement is made that, "A large percentage of pulmonary tuberculous cavities are 'tension cavities' and rarely close with thoracoplasty." Dr. Alexander referred to this statement, but I want to take exception to it also.

We have well over 1,000 thoracoplasty patients, and our percentage of cavity closures will run from 80 to 90 per cent. Almost any report on a group of thoracoplasties will be the same. *When the number of cavities closed with artificial pneumothorax and with phrenic* nerve interruption is added, there are not many left. I am afraid we are going to become tension-cavity conscious and neglect those procedures that have been effectual for so many years, for other measures more hazardous and less successful. I should stress this point as far as lobectomy is concerned. I agree with Dr. Alexander that we must remember that a small area of necrosis in a lung may be stretched by the intrapleural negative pressures to give the appearance of a large balloon cavity and a false impression of the actual amount of disease present.

It seems wrong to me to take out a large amount of good lung tissue because a small area of necrosis exists in it. This is frequently done in apparently blocked cavities.

As far as Dr. Head's paper is concerned, no one can take issue with any procedure in individuals with far-advanced, hopeless tuberculosis if it gives relief and prolongs life. I do not like the Monaldi operation but rather prefer cavernostomy in those patients in whom collapse therapy is impossible to accomplish. It often gives relief, and in some patients will cause cavity closure, but let us not go hog wild on tension cavities. There are not very many remaining after collapse methods are employed.

DR. LEO ELOESSER, San Francisco, Calif.—This difference of opinion merely shows us what I think we have been conscious of for many, many years, and that is that we do not know what we are doing when we are operating for pulmonary tuberculosis. I am merely going to discuss very briefly the pathogenesis of the tuberculous cavity and leave the question of treatment aside.

In the first place, I would like to protest at calling the cavities under discussion tension cavities. Dr. Vineberg and others have protested at my having called them blocked cavities. I think that protest is in order. If we will call them insufflation cavities, I think everyone will be satisfied. They are not tension cavities, to my idea, because "tension" connotes stretching. That is a literal translation of tension, and they are just the opposite of tension.

These insufflation cavities have two elements. They have in the first place the element of insufflation of the cavity itself. That insufflation element is not present all of the time. There is nothing, or very little, that works all of the time or 100 per cent of the time, as a very celebrated native of Illinois said a good many years ago. It is not necessary for a cavity to be under *constant* positive pressure in order to have it insufflated. All that is necessary is that it be under positive pressure more of the time than it is under negative pressure; this condition obtains in a great many tuberculous cavities, especially the round ones. What adds very greatly to the complication of the physics of these cavities is the extrinsic factor which Dr. Shipman has emphasized. These cavities are under influences not only from the inside, but from the outside, from influences that have to do with the relation of the caliber of the communicating bronchus of the cavity itself and of the bronchi that subserve the surrounding pulmonary parenchyma. And that brings us to a problem of physics that is so extremely complicated that I am afraid that I am not able even to state the questions that arise when one considers this complicated problem of aerodynamics.

It has been somewhat of a satisfaction to see how, since 1937, this topic has been elaborated. Some of the oldest inhabitants here, like Dr. Butler and others, will remember the development it has undergone, and that in 1937 so experienced a phthisiologist as our late president, Dr. Lord, stated that he had not seen enough patients with bronchial stenosis to be able to form an opinion concerning them. If, therefore, we will consider the insufflation cavity as one that is insufflated by pressures in which the positive exceeds the negative for a majority of the time, and if we will take account of the extrinsic factors, I think gradually, step by step, we shall arrive at some conclusion as to the pathogenesis of these cavities.

DR. VICTOR STRONG RANDOLPH, Phoenix, Ariz.—With Dr. O'Brien's permission I would like to mention a form of cavity drainage which I think should at least be brought to the attention of the Society. It was proposed in Toronto by Rogers and Shipman for use in post-thoracoplasty cavities, and I have used it in bilateral tuberculosis for patients in whom collapse measures cannot be used because they would so reduce vital capacity that life would not be possible to the patient, or at least not useful.

(Slide.) This was a man who was very sick. He had a large cavity in the top of the right lung and a good deal of disease in the balance of the lung. He had a small cavity in the top of the left lung and laryngeal tuberculosis; he wanted to get well. He was in bed for several months and not allowed to talk. A few months later the cavity in the right lung was a little larger and in the left lung, smaller; therefore, extrapleural pneumothorax was produced on the right side, and at a later time the cavity on the left side became very large, as can be seen, and therefore on this left side an Eloesser flap operation was performed. The next film shows that the cavity on the left side is gone, and this man, six years after his original illness, is well enough to work in a clerical capacity. This case illustrates the type of drainage that I wanted to show.

DR. LOUIS R. DAVIDSON, New York, N. Y.—I am not desirous of challenging the statement of anyone here. I do want to agree, and I do so heartily, with both Dr. Alexander and Dr. Eloesser. A few days ago, in this city, at a meeting of the American Surgical Association, Dr. McClure of the Ford Hospital of Detroit read a paper entitled, "Tannic Acid Treatment of Burns." This was an obsequy. He hoped that no one would again use tannic acid in the treatment of burns.

Regarding the Monaldi operation, as an individual procedure, I sincerely trust that we have read the obsequies. I think I am in a safe position to make this statement. Dr. Butler will tell us whether this society met in 1940 in Cleveland. At the meeting there, and all this is a matter of record, I deprecated the use of the Monaldi procedure as an individual method. The next year, in San Antonio, that statement was again made, and at this moment in no way do I feel that I want to change my opinion. I could give you my reasons for this conclusion, but I believe they are well known to you.

I had the privilege of knowing the late Dr. Pol Coryllos; I was perhaps affected by his dynamic personality and his fierce ambition. I am happy to find that today, many years after his death, pathogenesis, dynamics, and the closure of tension cavities are being discussed in almost his very words. He is now, perhaps, receiving commendations which he so well merited in this direction. To be sure, he and I disagreed very heartily, in some instances, on the question of the cavity in pulmonary tuberculosis. I agreed with him that there is a definite intracavitary influence at the bronchocavitary junction, but as Dr. Eloesser and Dr. Shipman have also stated, we insist that there are extracavitary influences. The Monaldi procedure in no way permanently influences these extracavitary factors.

I should like to show a few lantern slides. I devised this cannulating pulmonary cavity instrument some years ago in order to take frequent pressure readings while the cannula was in the lung and while we, with the aid of specially devised telescopes, could inspect the inside of these tuberculous pulmonary cavities. We know that the wall of the cavity is composed of three layers, the inner layer which is cheesy material, the middle layer or zone which is vascular in nature, and the outer or fibrous layer. This last zone is never noted upon inspection, except by the pathologist at the autopsy table. Through these telescopes we took colored pictures of the inside of the tuberculous cavity in the living patients. In only two of the thirty-seven cases studied could we definitely visualize the bronchus.

Here is a recess which is noted in all cavities. (Slide.)

I might say that almost every cavity which is cannulated by the Monaldi technique will close, but when the catheter or cannula is permanently removed, sooner or later, the cavity reopens.

Here is a patient with a catheter inserted into the cavity. You will note the fluid level. When this patient forcibly expired and the catheter was pinched, the cavity was obliterated. That is what occurs when good, constant negative pressure is applied to the cavity through the catheter, but when the catheter is removed, the cavity reopens, due particularly to extracavitary influence. This procedure may have a place, very occasionally, when it is combined with a thoracoplasty. With this in mind, an anterior thoracoplasty was first performed. The thought occurred to us that the brachial plexus and subclavian vessels might have dropped after the first rib had been removed, thereby leaving these structures without support and permitting them to fall over the space we would use for the cannulation. In order to determine this development, diodrast was injected into the cubital vein on the same side and the patient immediately x-rayed. It was found that these structures had not changed their original positions.

Because of this fact, the Monaldi was then performed, followed by an orthodox posterior thoracoplasty.

In closing, I may say the Monaldi procedure will not be used too often. While I do not call for obsequies, I feel that its reincarnation, in any form, should be very infrequent.

MAJOR WILLIAM W. WOODRUFF.—I wish to discuss Dr. Head's paper, as our experience, based on thirty-five cases, is similar to his.

We too have used Monaldi drainage almost entirely as a salvage procedure, reserving it for those cases in which other forms of therapy did not seem wise or possible. I believe it also has a place in the treatment of the insufflation cavities described by Dr. Eloesser, where in most instances it will be used in conjunction with thoracoplasty. When it is so used the question arises, should the Monaldi or the thoracoplasty be done first. There are disadvantages to each method. Our present procedure is giving reasonably good results and is as follows. Almost every cavity is packed because we have had the experience of finding a free pleural space at the time of introducing the trocar when none had been found by the aspirating needle. At the time of packing we have performed an anterior thoracoplasty, resecting portions of the first, second, and sometimes the third ribs. Then ten days to two weeks later the catheter is introduced. This method of procedure has the advantage that it is possible to keep the catheter in place while the posterior thoracoplasty is done and still take the entire remaining length of ribs without running a great chance of breaking through into the infected sinus tract.

Our indications and results in the thirty patients in whom the catheter was successfully introduced are as follows:

Seventeen had extensive bilateral disease. Of these seventeen, ten had either a cavity, unexpandable pneumothorax, empyema, or thoracoplasty on the opposite side.

Nine were chosen because of the acuteness of their disease.

The indication in the remaining four were age in two (58 and 60 years), refusal of thoracoplasty in one, and previous unsuccessful thoracoplasty in one.

I believe you will agree that any good results in this group can well be listed as salvage.

Now a word about our results in the group of thirty. In only four cases, or 13 per cent, have the catheters been withdrawn and the cavities remained closed without any other procedure being used.

Six of the patients, or 20 per cent, are dead. Four of the deaths were in the group of nine with acute tuberculosis. This is a high mortality rate, but an equal number (four) in the same group are much improved. At the time of interference it seemed as though very little could be done for any of them.

In eight cases, or 27 per cent, it became possible later to supplement the Monaldi by other procedures. Six patients have had thoracoplasty, one has had lobectomy, and one has had a cavernostomy.

Twelve, or 40 per cent, still have the catheter in situ. Some of these may eventually die, but in some the condition may eventually become arrested.

Based on this experience, we agree with Dr. Head that Monaldi drainage is a valuable procedure and enables us to salvage some patients for whom we have little else to offer.

Dr. Vineberg has been able to have suction throughout his hospital. We have not been so fortunate; our patients have been scattered throughout the village, making necessary to have individual suction for them. We have developed a simple, portable cabinet unit. Suction is obtained by using a Stedman suprapubic suction pump. The pressure is controlled by using a mercury safety valve, and an anaeroid pressure gauge from a Zavod pneumothorax machine has been connected in the circuit so that the pressure fluctuation may be constantly observed. It is simple, reliable, and safe. The motor runs continuously and we are able to maintain a constant predetermining negative pressure.

DR. OTTO C. BRANTIGAN, Baltimore, Md.—I wish to emphasize that tuberculous pulmonary disease is a dynamic process. The mechanism which produces a tension cavity is not always constant or permanent. The intracavitary pressure will vary from time to time, fluctuating between positive and negative pressures. It would be ideal if collapse therapy could be applied during the phase of negative pressure within the cavity.

DR. FRASER B. GURD, Montreal, Canada.—Just a word or two with reference to Dr. Alexander's discussion regarding the contribution of Dr. Vineberg and Dr. Kunstler. He referred to the fact that in the article published by them in *Surgery, Gynecology, and Obstetrics* in March of this year, the total number of cavities obliterated in the group subjected to transthoracic intracavity drainage is small. I wish to point out that this group of cases refers only to thoracoplasties that have been done upon chests in which the tuberculous cavities have been one inch, or more, in diameter and that the more simple lesions which we prefer to treat are not included in this group. In other words, those patients who are not in the salvage group are not included in these figures. I am not personally able to state offhand the number of straightforward cases in which operation has been done, but in those patients who had smaller cavities and who were not needled, the number of closures was very high and would have raised the total number of successful cases substantially.

Dr. Vineberg and Dr. Kunstler are not, I believe, suggesting that the problem of obliteration of large cavities, whether these be positive pressure or not, has been solved. In this attitude every one of us in this room would be in agreement; Dr. Vineberg and Dr. Kunstler have, however, brought forward for consideration a method of investigation which seems to be safe and, in the main, to be very satisfactory. I know that I personally, watching the work of these younger men, have been fascinated by the information which they have obtained and pleased by the very substantial improvement of the results in our clinic since routine determination of pressure in all larger cavities has been carried out.

DR. ARTHUR VINEBERG, Montreal, Canada.—The first thing I would like to say is that we have never, even in our writings, suggested that intracavity suction drainage was a means alone of closing a cavity of the tension or positive pressure type. You speak of intracavity or extracavity factors. We, too, believe in the extracavity factors, and that is why we believe in doing an anterior stage thoracoplasty.

With regard to Dr. Alexander's remarks—it is quite true according to our figures that we did have 42 per cent of cavities which were needled and proved to be tension cavities. Ordinarily across the country, about 30 per cent of cavities failed to close after thoracoplasty and became residual cavities, that is if only cavities are considered. In reporting 1,500 patients treated by thoracoplasty with 83 per cent closure of the cavities, I would like to go over those cases and see how many of them were simply minimal disease or small, tiny interloculated cavities. We are talking about cavities all of which are larger than 2.5 cm. This is the first point.

The next point is that, I quite agree with Dr. Alexander that some of these patients may have tension cavities at the apex when we needle them. But in a certain percentage of these cavities following thoracoplasty, the bronchus becomes twisted and closed off, which would help to explain the discrepancy between the figures of 42 per cent tension cavities and 17 per cent failure of cavities to close by thoracoplasty.

Dr. O'Brien objects to needling of cavities. We have had absolutely no complication from needling in our series. We think that it is a crime to make a respiratory cripple of a man because he has one cavity. It is not the right thing to do a seven-rib thoracoplasty on a man if a five-rib operation following intracavitary suction drainage will suffice. A good healthy lung is being obliterated instead of the diseased area. We are attempting to knock out only the diseased areas and not sacrifice healthy lung in order to do so.

Dr. Davidson showed us where he had examined various cavities from bronchi and found only two bronchi in the endoscopic examination. May I point out to Dr. Davidson that the average pathologist who examines the lung does not find the tension or at least the valvular mechanism because he doesn't look for it, and we, ourselves, have gone back over cavities which were declared to be without any definite valvular mechanism in their draining bronchi, and we have found in every case a bronchus leading to the cavity with a trap-door mechanism or a bulbous type of swelling in the bronchial wall. You have to look for it, and you have to look for it at the autopsy table. It will not be seen necessarily through the endoscope.

In closing, with regard to Dr. Head and Dr. Woodruff who suggest that this method should be reserved for salvage cases, it seems to me that their cases are no criteria whatsoever of the efficiency of this method. The previously mentioned workers have attempted to salvage people. There is no point in trying to salvage patients with a vital capacity of 1,000 c.c. or less, but if such an attempt is made, from the results of Dr. Head's cases and from what Dr. Woodruff reported, I would say that it speaks very well for intracavity drainage per se. Why reserve this method for closing cavities in salvage cases? Why not try to close large cavities with the minimum amount of damage to healthy lung?

DR. JEROME R. HEAD, Chicago, Ill.—I wish to express my appreciation of the very excellent work that Dr. Riggins and Dr. Vineberg have done. I believe that it is important and will lead to improved practice. I also would express my agreement with what Dr. Alexander and Dr. O'Brien said. I believe that the truth is neither here nor there. I am sorry that Dr. Davidson did not say that Monaldi suction should be abandoned completely. It would have served as a text for a lecture. Throughout the whole history of collapse therapy and tuberculosis we have had many new techniques introduced. Eventually someone says that he has given up this or that operation as being of no value. I have found that everything that has been added—paraffin pack, extrapleural pneumothorax, Monaldi suction—each has a place; there are patients who cannot be treated in any other way. The one who practices the best will use them all as they are indicated.

obstruction with resultant anoxia. Bronchial râles have been present in all patients. In a poorly emptied tracheobronchial tree, bronchial râles persist in spite of cough and expectoration. When they are wheezing and high pitched, they have a dry quality. They may be loud enough to be audible at the bedside and in such instances the patient appears to be having a typical asthmatic attack. In spite of the dry quality of these râles, they have disappeared after the patient has commenced raising tenacious sputum. In other patients, bubbling sonorous râles occasionally predominate with an accompanying classical tracheal rattle. When râles of this sort are present the sputum raised is frequently of a more liquid character.

Distant bronchial breathing may be heard over small areas, usually signifying patchy lobular atelectasis, but of course it may represent a pneumonic process. The diagnosis is certain if the intensity of the bronchial breathing or its location changes with forced cough.

Fever and toxicity are constantly present. While at this stage early bronchial infection may be a minor factor, the fallacy of treating all of these patients for a bronchopneumonia is obvious. The history, the constant wet cough, and widespread bronchial râles are signposts that will not be ignored in the proper evaluation of the patient's condition.

ROENTGENOGRAPHIC FINDINGS

The frequent coexistence of an extensive thoracic wall, pleural or intrapulmonary lesion makes the shadows possibly due to partial or complete bronchial obstruction very difficult to interpret. On the other hand, a considerable degree of obstruction may be present without roentgenographic evidence. When any question arises as to the diagnosis, more weight should be given to the clinical picture. Whenever possible, roentgenograms in at least two planes should be taken. In certain specific instances, collateral information may be gained by thoracentesis when it is considered in the light of suggestive roentgenographic findings. The shadows caused by atelectasis must be differentiated from those produced by pulmonary contusion or hematoma. With intrapulmonary bleeding the shadows are quite likely to be round or oval and in the case of a hematoma there may be small, loculated airpockets present. Intrapulmonary bleeding will not result per se in cardiac or mediastinal shift.

TREATMENT

With any degree of bronchial obstruction, treatment is aimed at improving the impaired tracheobronchial drainage as rapidly as possible. Cardiorespiratory imbalance from mediastinal shift, shock, ileus, etc., is corrected simultaneously. Thoracentesis is an extremely valuable preliminary step in the treatment of bronchial obstruction, and must always be performed at once whenever there is any evidence of pleural fluid. Gastric and intestinal decompression should be commenced as indicated. The development of deep full respirations and an efficient cough is mandatory.

Pain from thoracic wall contusions or from fractured ribs has been a frequent reason for inability to breathe deeply and to cough and raise sputum. In treating a large number of thoracic casualties one of the most significant observations has been complete confirmation of the value of intercostal and paravertebral nerve block in the control of thoracic wall pain. This has been reported previously by Rovenstine and Byrd,[7] although these authors did not elaborate on the relationship of thoracic wall pain to wet lung and defective bronchial drainage. The indications, technique, and results of this simple and valuable procedure have been given.[10] Nerve block has completely relieved pain and discomfort for a period of time averaging not less than twenty-four hours. But it may be that the relaxation of voluntary muscle in the thoracic wall is important in the relief of pain well beyond the time of action of the anesthetic agent. With the exception of severe flail chest, nerve block has precluded all necessity for strapping the thorax, in itself a procedure which decreases respiratory exchange and favors the retention of bronchial secretions. Nerve block is, then, indicated at once on any patient with faulty bronchopulmonary elimination who is complaining of thoracic pain. It should be used prophylactically in all patients with severely painful chests, where respiration and cough are limited. The cough almost immediately becomes efficient and productive; dramatic improvement usually follows within one hour. Nerve block may be the only procedure necessary to aid in clearing the obstructed bronchi (Case 3).

Carbon dioxide inhalations combined with the frequent changes of the patient's position are likewise of considerable value. The gravity effect of positional change on bronchial secretions is too well known to require further description. We much prefer 100 per cent CO_2 when it is available, administering it for two or three deep breaths by means of a catheter and small funnel. Forced coughing, during and following the brief stage of hyperpnea, is of the utmost importance, a detail not often sufficiently stressed. Without forced cough, the administration of CO_2 loses much of its effectiveness. When no CO_2 is available, rebreathing into a paper bag may be substituted. The value of carbon dioxide is twofold; the gas itself is a true expectorant[3] and, mechanically, hyperventilation loosens and propels viscid secretions along the tracheobronchial lumen.

CATHETER SUCTION

Depending on the condition of the patient, on his roentgenographic findings, and on our clinical judgment as to the progress toward clearing, the measures just enumerated are continued for from six to twenty-four hours. If the patient does not progressively improve or if there is evidence at any time of further blockage, mechanical aspiration of the tracheobronchial lumen is resorted to without delay. This is performed by either of two methods: tracheobronchial catheter aspiration[4] or bronchoscopy. Each method has its advantages and there are but few clear-cut indications for the use of one rather than the other method. Bronchoscopy may be followed by repeated catheter aspirations. In both methods the basic aims are the removal of excess fluid in the lower airway, the loosening of deeper secretions by local manipulation of the tracheobronchial tree, and the development of a more efficient cough.

The equipment for catheter suction is almost always available and the technique is simple. The fact that with practice no anesthesia is necessary makes this method one that can most easily be used in an emergency. Rarely, a small amount of topical anesthesia may be required if a hyperactive gag reflex is present. The technique should be mastered by surgeon and internist alike; many lives will be saved thereby. A source of suction delivering from fifteen to twenty pounds negative pressure (ordinary ''nose and throat'' suction machine), connecting tubing, and a No. 16-18 new urethral catheter are all the materials necessary. A portable hand-driven suction machine has been designed by one of us (L.A.B.)[5] for use when no source of electricity is available. The Robinson type of urethral catheter with two openings is preferred. The patient is placed in semi-Fowler's position, the neck is flexed, and the tongue pulled rather sharply forward to anchor the larynx. If the patient is unconscious, the neck can be flexed by raising the head on a pillow or folded blanket. Without suction, the catheter is passed through the more widely patent nostril and advanced until the larynx is reached. The catheter is then withdrawn for about 1 cm. and the patient asked to take a rapid, deep breath. During inspiration, the catheter is quickly advanced into the trachea. Success in the manuever will be signified by coughing, passage of air through the tube, and huskiness of the voice. Occasionally it will be better to advance the catheter during the expiratory phase of a cough. After the catheter has been advanced for several centimeters into the trachea, suction is commenced. The patient is urged to cough and usually responds violently due to contact with the tracheal mucosa. The catheter is slowly advanced and retraced to provoke coughing and prevent excessive suction on the mucosa in any one spot. Suction is stopped after about five seconds and the patient allowed to take two or three deep breaths so that not all the air will be drawn out of the bronchial tree. Aspiration is then commenced again and after the trachea is dry, attention is turned to the main bronchi. The patient's head is turned slightly to the left in order that the right stem bronchus may be entered. It is somewhat more difficult to enter the left stem bronchus; the patient's head must be turned far to the right and the chin somewhat elevated. The catheter should be advanced as far as possible into each stem bronchus. Frequently, the patient is rolled on his side with the more involved lung uppermost, so that dependency will aid in draining the smaller bronchi. The continuous periods of suction should not be longer than five seconds and the patient should not be allowed to become exhausted or cyanotic. Following catheter suction the patient coughs with greater efficiency because of increased ventilation. He should be watched closely, however, for further accumulation of secretions. Since there is no trauma to the larynx or bronchi, catheter aspiration may be repeated at two- to three-hour intervals.

Certain specific uses of catheter aspiration should be listed. Acute emergencies on the ward should undoubtedly be treated by this means. When an active traumatic thoracic service is in operation it is wise to have a tracheobronchial catheter aspiration set prepared on the ward at all times. In the unconscious patient where it is probable that frequent aspirations will be necessary, catheter suction should probably be performed from the beginning. Occasionally there is the desperately ill but conscious patient in whom tracheobronchial cleansing is urgently needed, but even the manipulation necessary for a bronchoscopy may prove hazardous; catheter aspiration may well prove a lifesaving procedure. Postoperatively, catheter aspiration is of great value if there is any question of retained secretions. Rarely, it may not be possible to pass the catheter because of an unusually flaccid epiglottis. Technically, there is no other condition which should prevent the passage of a catheter.

BRONCHOSCOPY

Bronchoscopy is a highly technical procedure to be performed by someone especially trained in this technique. General anesthesia is contraindicated in the presence of excess bronchial secretions so that topical anesthesia must be employed. Bronchoscopic aspiration is a more efficient procedure than catheter suction. Since it is done under direct vision, both stem bronchi will be aspirated in a more efficient manner than catheter suction. With the use of curved suction tips, more adequate drainage of the smaller bronchi can be accomplished. Further increase in the airway is provided by mucosal shrinkage with pontocaine (or cocaine) and epinephrine, which procedure is not possible with catheter suction.

Under certain conditions bronchoscopy is carried out at once without waiting for a trial of more conservative measures. Bronchoscopy is the treatment of choice when the obstruction is due to particulate material, blood clots, and thick mucous plugs.[5] In bilateral atelectasis the patient has a sufficient reduction in his vital capacity so that a curative procedure is indicated without delay (Case 2). In unilateral massive collapse, immediate bronchoscopic aspiration is preferable to a more conservative regimen. With an already reduced vital capacity, the sudden loosening of large amounts of secretion from the obstructed bronchus can well flood the normal bronchus with grave results. In partial tracheal obstruction an immediate bronchoscopy must be considered. Here again, there is great danger of aspirating excess secretion into the more normal bronchus. When there is sudden recurrence of obstruction after partial or complete clearing of the bronchial lumen, early bronchoscopy is demanded. At times a patient is admitted to the ward completely exhausted, without the strength to cough and with the tracheobronchial lumen brimming with secretions. Immediate aspiration is mandatory. Here is a seeming paradox, that the sicker the patient, the more necessary is an immediate bronchoscopy (Case 1). Failure to follow this principle will result in disaster. Stomach secretions have an early deleterious effect on the tracheobronchial mucosa.[6] When an unconscious patient aspirates vomitus it is an imperative indication for mechanical cleansing of the tracheobronchial tree before the patient leaves the operating room. Bronchoscopy is much the preferable procedure. Catheter aspiration must, however, be substituted if the patient has only partially reacted from the anesthesia and his neck and jaw muscles have become tight. Justification for stressing this point lies in the fact that much of the forward surgery is of an emergency nature. Postoperative vomiting will, therefore, be a more frequent complication than in civilian surgery.

CASE REPORTS

Many patients showed a significant degree of intrabronchial obstruction following thoracic trauma. Three case histories have been chosen from this group to illustrate many of the problems of diagnosis and treatment. Patients were received at the thoracic surgery center from four to five days after injury during the Sicilian invasion and the early part of the Italian Campaign.

CASE 1.—A 23-year-old soldier received multiple machine gun bullet wounds while fighting in Sicily on July 15, 1943. The most serious injury was a right-sided thoracoabdominal wound, the missile entering the thorax anteriorly just below the clavicle and coming out through the upper abdominal wall. An emergency laparotomy was performed at a field hospital. A laceration in the right dome of the liver was found and packed. At the conclusion of the operation the patient was in critical condition from right-sided tension hemopneumothorax and an obstructed lower airway. Twenty-two hundred cubic centimeters of blood and air were removed from the right pleural cavity, followed at once by bronchoscopic aspiration of obstructing secretions. There was immediate dramatic improvement. The tension pneumothorax was treated by an indwelling needle attached to a waterseal bottle. This needle did not function during transportation. The patient arrived at the thoracic surgery center on July 19 in poor condition with dyspnea, cyanosis, and a weak rapid pulse. Expectoration of tenacious bloodstreaked secretion eased the cyanosis somewhat. The patient had a wet cough and raised sputum constantly. There were coarse râles over the trachea and parasternally on the right. Thoracic roentgenograms showed a right-sided patchy obscuration apparently due to a small amount of pleural fluid and, perhaps, scattered areas of atelectasis. On thoracentesis, only a few cubic centimeters of thin bloody fluid without odor were obtained. Immediate bronchoscopy was productive of much bloody tenacious secretion coming from the trachea and right stem bronchus. The patient raised several ounces of similar material during the next three hours and his condition improved. On July 21, the patient again experienced acute obstructive respiratory difficulty, and emergency tracheobronchial catheter aspiration *on the ward* was undoubtedly lifesaving. This material was bloodstreaked and had a faintly foul odor. Roentgenograms were repeated and the obscuration in the right upper part of the thorax had increased. Thoracentesis now revealed thin bloody foul pleural fluid, identical to that which had been aspirated. Thus, the existence of a bronchopleural fistula was established even though the roentgenogram disclosed neither fluid level nor mediastinal shift. Intercostal water-seal catheter drainage was immediately performed and there was complete loss of the tracheobronchial wetness. Several days later, open thoracostomy with rib resection gave adequate drainage. The fistula closed spontaneously and the empyema cavity eventually became obliterated.

On three occasions bronchial aspiration was a lifesaving procedure for this soldier. We are convinced that tracheobronchial obstruction may often be a factor in early death from thoracic injury. Many of these deaths can be prevented if the possibility of bronchial obstruction is not overlooked. Another feature worthy of comment in this patient is the dramatic fashion in which proper drainage relieved bronchopleural spillage as in any case where a fistula connects a hemothorax or an empyema with the tracheobronchial tree.

CASE 2.—A 23-year-old British soldier was wounded in action on the Salerno Beachhead on Sept. 9, 1943. He suffered a perforating gunshot wound of the right side of the chest. The patient's wound was dressed on the beach and he was evacuated to a Landing

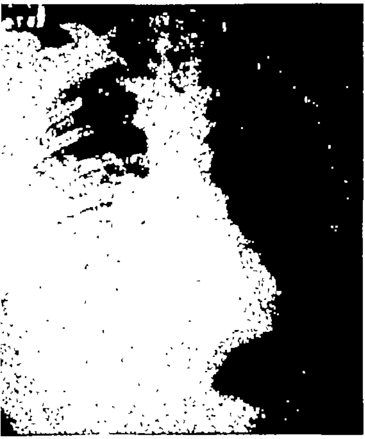

Fig. 1.

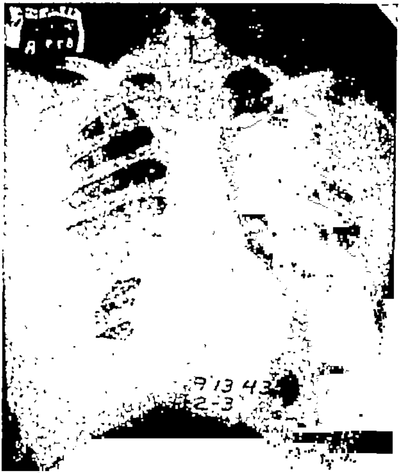

Fig. 2.

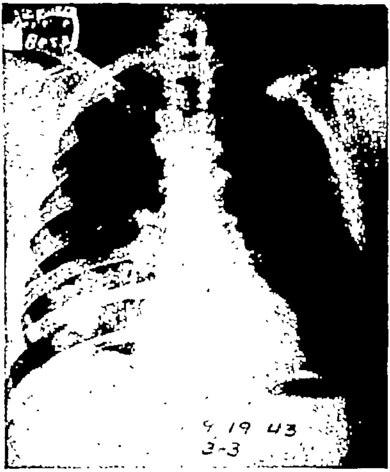

Fig. 3.

Fig. 1.—Bilateral basal atelectasis; patchy pulmonary contusion of the right side.

Fig. 2.—Sixteen hours following bronchoscopic aspiration; the atelectasis has cleared completely.

Fig. 3.—Six days following bronchoscopy; rapid clearing of the area of intrapulmonary bleeding.

Ship Tank (LST) on which he remained for three days, arriving at the thoracic surgery center on September 12. He was exhausted, dehydrated, and dyspneic and he had a wet cough without expulsive force. He was raising only small amounts of faintly bloody mucopurulent sputum. Physical examination showed no shift of the mediastinum. Coarse rhonchi were heard over the midline. At the base, bilaterally, were areas of faint bronchial breathing associated with fine râles, which shifted slightly in location and intensity following cough. Roentgenograms (Fig. 1) showed a bilateral basal triangular shadow extending down and out from the hilum and considered typical of bilateral atelectasis. Peripherally, at the right base, the rounded area was interpreted as probable intra-alveolar bleeding (pulmonary contusion).

Bronchoscopy was performed several hours after admission because of the patient's extensive bronchial occlusion, lowered vital capacity, inability to cough efficiently, and exhaustion. Obstructing tenacious mucosanguineous secretions were aspirated from both lower lobar bronchi. After mucosal shrinkage, the patient coughed up quantities of tiny pluglike casts composed both of blood and of mucoid material. These obviously had been obstructing small bronchi bilaterally. The patient was immediately relieved of the dyspnea and the breath sounds at the base lost their bronchial quality. The cough was productive of approximately eight ounces of sputum during the next eighteen hours. At this time roentgenograms showed complete clearing of the bilateral atelectasis (Fig. 2). Progressive clearing of the area of contusion occurred in the next six days (Fig. 3).

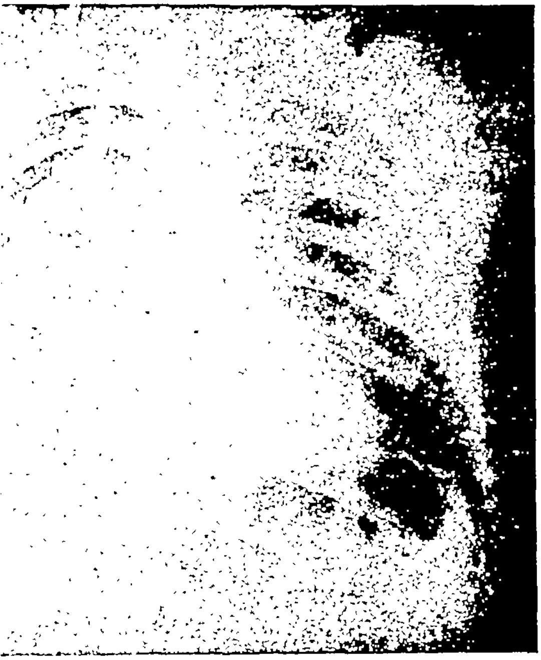

Fig. 4.

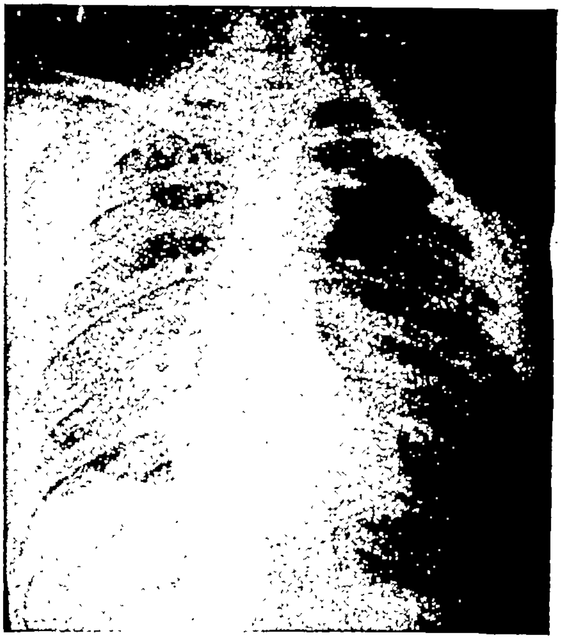

Fig. 5.

Fig. 4.—Right-sided hemopneumothorax; probable partial bronchial obstruction: the seventh and eighth ribs have been fractured posteriorly.

Fig. 5.—Extensive clearing twenty-four hours later.

Case 3.—A 27-year-old soldier was wounded in action in Italy, on Sept. 13, 1943. He suffered a perforating "sucking" wound of the right posterior thoracic wall. Immediate treatment consisted of a massive occlusive dressing and plasma by vein. Within twenty-four hours the patient had been admitted to a semimobile evacuation hospital. Operation consisted of wound trimming, removal of rib fragments, repair of pleural defect, and tight skin closure. The patient was having constant hemoptysis at this time. Because of tension pneumothorax an indwelling needle with flutter valve was inserted in the second anterior interspace. It was removed in seventy-two hours. The patient arrived at the thoracic surgery center on the evening of September 18. He was in no respiratory distress and was only slightly dyspneic on exertion. Physical examination showed a right-sided hemopneumothorax without mediastinal shift. The wounds were clean. Early next morning the patient suddenly developed great respiratory distress. He commenced to perspire, became moderately cyanotic, and developed a constant wet, painful cough, productive of small amounts of tenacious mucoid sputum. The pain was limited to the posterior thoracic wall on the right. Loud coarse rhonchi were heard over both lung fields posteriorly. Roentgenograms, taken immediately, showed a hemopneumothorax on the right with approximately 30 per cent compression of the lung (Fig. 4). In addition, there were patchy areas of pulmonary obscuration which were difficult to separate from the shadows due to the intrapleural fluid. Because of the patient's obvious distress and the bronchial obstruction, it was tentatively decided that bronchoscopy should be done. First, however, right posterior intercostal nerve block was performed, from the fifth to the tenth thoracic vertebrae inclusive, and 1,200 c.c. of bloody fluid and air were removed from the pleural cavity. Thus, pain and the splinting effect of pleural fluid were removed. Almost immediately the patient was able to breathe more deeply and to cough effectively. Within a short time after productive cough started, the patient stopped perspiring and the bronchial râles disappeared. He raised six ounces of extremely tenacious mucoid sputum during the next five hours. The efficient productive cough, aided by small amounts of CO_2 continued for three days and gradually subsided. On September 20, thoracentesis was productive of 60 c.c. of old blood and air. Shortly thereafter a roentgenogram showed remarkable clearing both of the pleural cavity and of the pulmonary parenchyma (Fig. 5). The intrathoracic abnormalities disappeared completely in a few days. On the eleventh day after injury, skin separation of the wounds occurred because of an extensive deep serum collection. The pleural closure remained tight.

DISCUSSION

The tracheobronchial tree has as its primary function that of providing an airway from the alveolus to the outside atmosphere to permit the exchange of gases necessary for the maintenance of life. However, one must remember that it has other important functions as well. There is also a definite secretory function and the capacity to rid itself of the secretions, other fluid substances, and aspirated material that may be abnormally present within its lumen. The action of the cilia and the peristalsis of the bronchial musculature effect this cleansing of the bronchial tree. These intrinsic actions of the bronchi and trachea are aided by the voluntary cough mechanism. Little is known about the effect of the chest trauma on ciliary action of the mucosa and bronchial peristalsis. One might presume, however, that these functions would not be normally active. Nevertheless, there is definite clinical evidence that the normal cough mechanism is seriously impaired when there is even moderate thoracic trauma. Thus, the interference with the emptying of stagnated fluid substances in the bronchial tree results in bronchial obstruction, which if unrelieved may be followed by pulmonary atelectasis and pneumonia. With this concept of the function of the bronchi and the derangements thus brought about by thoracic trauma, the treatment then becomes simple and logical. The measures advocated are mainly those to improve inadequate tracheobronchial drainage.

It is probable that varying degrees of pulmonary atelectasis may be present early in these patients, although the diagnosis cannot be made early in most instances. This is due to several reasons. The evidence of large amounts of fluid in the pulmonary tree, shock, severe dyspnea, and anoxia dominate the clinical picture soon after injury. Furthermore, one is forced to proceed, vigorously treating the patient without waiting for a roentgenographic examination. In fact, therapeutic measures must be employed to make the patient fit for x-ray examination. We have suspected pulmonary atelectasis early in these patients but have not had a clinical picture nor the confirmatory roentgenographic evidence. Vigorous treatment was instituted promptly on the day of injury and the days immediately following injury, without resorting to x-ray studies. Although we have suspected atelectasis soon after injury, actually the diagnosis has been made with certainty a few days after the wounding. The question of just when pulmonary atelectasis first develops in these cases will be solved when it is possible to take serial portable chest x-rays. The progress of the patient should be plotted soon after the initial trauma and at hourly intervals if necessary. However, in the forward medical installations where such facilities are not available this question is of academic importance, if one proceeds, vigorously treating the patient according to the principles that have been outlined.

CONCLUSIONS

1. Bronchial obstruction may be present in patients with chest injuries as a result of retained fluid substances in the pulmonary tree.
2. Persistent bronchial obstruction is an important factor in the development of pulmonary atelectasis and pneumonitis following thoracic trauma.
3. The principles of improving inadequate tracheobronchial drainage are discussed.
4. Illustrative case histories are presented.

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CERVICOMEDIASTINAL LYMPHANGIOMA WITH CHYLOTHORAX

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THE occurrence of chylous effusions in the chest and abdomen has been noted sporadically in the literature over the past 300 years. Their rarity has stimulated authors to report individual experiences. Recently these reports have been of more significance because of various therapeutic measures now being instituted in attempts to save the lives of patients with chylous effusions. The case to be presented does not offer a new therapeutic problem but warrants report because of the unusual nature of the basic lesion and because of certain implications which may aid in the clarification of some previously unexplained cases.

In a consideration of chylothorax, two major groups of etiologies, the traumatic and nontraumatic, are unrelated. The cases due to trauma constitute an interesting subject in many respects, such as the numerous ways in which trauma can give rise to extravasated chyle and the problems of management which arise. They are not the subject of the paper, but, for the sake of some completeness, a few of the salient points are worthy of note.

The traumatic cases present a rather characteristic sequence of events after the precipitating injury. There is usually a quiescent period (from four to six days up to two months) following the injury before pulmonary symptoms appear. This free period is more or less suddenly interrupted by the onset of dyspnea, often a severe shocklike picture, and frequently cyanosis. Chest pain may or may not be a feature. On examination, the patient will be found to have a massive pleural effusion on one or possibly both sides, and aspiration then establishes the diagnosis with the removal of a milky fluid. When sufficient fluid is removed, dyspnea and cyanosis are relieved and the patient is quite comfortable. Then the problem of management arises because the effusion generally reaccumulates rapidly, again causing respiratory embarrassment. With slow leaks requiring only infrequent aspirations, the patient may carry on for weeks or months. But if the accumulation is rapid and persistent, dehydration and emaciation are likely to develop and death may occur in a few days to a few weeks. The most serious loss is that of proteins in the chyle, and measures to diminish this must be instituted.

The types of trauma which have been responsible for chylothorax include hyperextension injuries, penetrating wounds, fractured ribs and vertebrae, and accidental lacerations occurring in the course of surgical procedures. Other traumatic types may result from coughing spells, convulsions, blast injuries, and injury to infants at the time of delivery or shortly thereafter. The most common type is the hyperextension injury from falls, bruises, etc., in which the thoracic spine is suddenly and acutely extended. Under these conditions the right crus

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of the diaphragm is said to compress the rather fragile thoracic duct against the vertebrae, causing a laceration. The chyle then leaks into the mediastinal soft tissues (or lower, giving rise to chylous ascites) in a retropleural position. A large collection of chyle gradually accumulates here until a rupture through the parietal pleura occurs due to pressure or tension. The sudden release of all this fluid compromises lung and mediastinum and produces the clinical picture to which reference has been made. It would not occur if an intact pleura were present as might happen in penetrating wounds or fractured ribs. This fact has been established by the demonstration of "retropleural chylomas" in the mediastinal tissues at operation, at post-mortem examinations and by x-ray examination.[2, 9, 11, 12] Brown in 1937 proposed drainage of these chylomas through a paravertebral, extrapleural approach to obviate pulmonary complications.[12]

Traumatic chylothorax occurs most frequently on the right side due to the anatomy of the thoracic duct (which arises from the cisterna chyli to the right of the midline and opposite the body of the second lumbar vertebra). It enters the thorax to the right of the midline, passes beneath the right crus of the diaphragm and maintains this position in the posterior mediastinum up to the fourth thoracic vertebra, where it courses to the left side and ascends into the root of the neck for a short distance, bends and enters into the left subclavian vein at the angle between it and the internal jugular vein. This termination is not constant and may consist of several branches, all entering this vein or some going also to the left internal jugular or the right subclavian vein, or both.[1]

Therapeutic measures have been conservative in the main. These consist of aspirations of fluid as often as is necessary, depending on the patient's respiratory status; low fat, high carbohydrate and protein diets; or nothing by mouth with intravenous fluids only for a time. More recently, patients have been treated with intravenous infusion of some of the chyle in an attempt to offset the great loss of proteins, particularly in prolonged leakage. Anticoagulants have been used (citrate and heparin), and with bacteriologic controls this material has been infused even when four days old without reaction. Some untoward reactions have occurred, however, and at least one death is recorded, due presumably to anaphylaxis.[7] Plasma and blood transfusions are valuable in maintaining fluid equilibrium. Other measures have included pneumothorax after aspiration, phrenicectomy, external drainage, and injection of irritating solutions into the pleural cavity to promote adhesions.

From experimental work it is known that there is a large collateral bed for the thoracic duct circulation; hence, ligation of the leaking duct would seem to offer a hopeful possibility. However, even ligation of both ends after surgical trauma failed to prevent chylothorax in a recent patient with trauma.[7] One can assume that ligation in patients with trauma is too sudden for collateralization because, at autopsies revealing generalized malignant metastases, extensive blockage of the thoracic duct and many of its branches has been found without chyle leakage.

Mortality rates average 50 per cent for the patients with conservative treatment. Surgical intervention, such as ligation and repair attempts, produces a higher rate—up to 100 per cent, according to one article.[9] Operative leakage is attended with a 10 per cent mortality, and in eight bullet wound cases a 25 per cent mortality is reported.[13]

The subject of nontraumatic chylothorax has been thoroughly presented by Yater,[1] who found about 100 cases mentioned before 1935. The most common etiology is metastatic malignant disease which gives rise to obstruction to the duct and/or its radicles by implants within the lumen or by pressure on the duct from adjacent nodes in the supraclavicular area. There are included abdominal carcinomas, lymphosarcoma, and Hodgkin's disease. Tuberculosis causing obstruction of the thoracic duct is next in importance and is most common in children in whom pressure by lymph nodes or intrinsic obstruction is the cause of leakage. Other miscellaneous causes listed by Yater included lymphangiectasis, localized thrombosis of the subclavian vein in the region of its junction with the duct, filarial obstruction, and erosion by aneurysms.

Fehr[1] proposed the theory that slow obstruction would not cause an effusion unless many of the collaterals are likewise blocked (by carcinoma for instance). The reason chylous effusion is not seen more often in terminal malignancy is that death no doubt occurs before obstruction of this extent can develop.

Prognosis in patients with nontraumatic chylothorax depends on the nature of the underlying disease and the influence of therapeutic measures, such as radiation for lymphoblastomas.

Some interesting points about chyle may be mentioned. In the past, considerable effort was made to distinguish between true chylous effusions and so-called pseudochylous fluid. The latter was supposed to be milky fluid resulting from alterations in long-standing effusions in body cavities associated with malignancies, tuberculosis, cirrhosis and nephritis. The color change was said to be due to the appearance of a lecithin-globulin combination, and by means of laboratory tests the two fluids could be distinguished.[35] The main differences were the absence of fat and a specific gravity below 1.012 in pseudochylous fluids. More recently the existence of pseudochyle has been questioned.

The finding of fat in an effusion is now considered to be evidence of its chylous nature. Brescia,[4] quoting from Gandin, states that the milky appearance is due to the presence of emulsified fat and that chyle is the only source of such emulsified fat. This would appear to hold despite an observation attributed by Gerster[23] to Henschen to the effect that chyle and lymph are very similar in a starving animal and that lymph after fatty meals (for example, lymph from an extremity) contains fat in practically the same percentage as chyle-bearing lymphatics.

The nature of chyle with its fat content makes the staining reaction with sudan III of diagnostic importance. In our case and in several cases in the literature, the chyle did not stain. This does not rule it out as chyle but does require explanation. Sudan III does not stain neutral fat globules which often constitute from 70 to 87 per cent of the total lipids of the chyle of experimental animals.[17] Thus the constituents which should react may not be in a concentration sufficient to be detected. Briefly then, chyle has a milky appearance, has a specific gravity of over 1.012, contains emulsified fat which usually can be recognized with sudan III, and is sterile, and, if the fluid is alkalinized and shaken with ether, it will clear.

Cytologic studies of the chylous fluids and of peripheral blood smears have given some interesting findings.[17] Thirteen peripheral blood studies on ten different patients reported on in the literature showed six to have a high percentage of lymphocytes and seven to have a range of from 6 to 16 per cent. The cells in chyle have consistently shown a high percentage of lymphocytes, and experimentally this was confirmed by average cell counts of 20,700 per cubic centimeter, with from 85 to 90 per cent lymphocytes. Along with these findings, peripheral blood smears after ligation of the thoracic duct revealed a drop in the number of lymphocytes and eosinophiles in spite of the occurrence of a leucocytosis. Finally, it was noted that the milkiness of chyle varied directly with its fat content.

It is stated in numerous articles that the average rate of flow of chyle in the thoracic duct is from about 130 to 195 c.c. per hour, with marked variations occurring at mealtime.[1] Chyle is a mixture of liver and intestinal lymph with variable protein and fat content, depending on the relative mixture of high protein liver lymph with the bowel drainage which contains a variable concentration of fats. The thoracic duct is equipped with a valve near the subclavian junction which opens toward the vein. It has been observed that chyle has a rhythmic variation in its flow, the result of numerous factors, including intestinal contractions, contractions of the cisterna chyli and the thoracic musculature and the movements of respiration.[4] Experimental ligation of the thoracic duct in dogs rarely causes chylothorax. Only after the superior vena cava was ligated could chylothorax be produced in 50 per cent of the animals.[1]

It has been mentioned that chyle is strongly bactericidal. Furthermore, it is frequently observed that chyle can remain at room temperature for weeks without putrefaction. It can therefore be assumed that infection of chylous effusions will occur rarely and that appropriate surgical efforts to solve the problem can be contemplated in patients with traumatic cases without fear of infection.

CASE REPORT

G. C. (No. 516387), a 32-year-old man, was admitted to the Mount Sinai Hospital for the first time Feb. 8, 1944, giving a past history of episodes of crampy abdominal pains for the past ten years associated with dietary indiscretions. The last attack was from four to five months before admission, at which time pain was almost immediately relieved by medication. Four years before, gastrointestinal films at another hospital were reported to be negative.

Three weeks before admission he developed an upper respiratory infection characterized by cough with very little sputum, general malaise and fever. He was considered to have a virus infection, possibly a virus pneumonia.

Two days before admission he noted a swelling in the left supraclavicular area without local pain or tenderness. That evening he developed generalized abdominal pain, most marked on the left side. The next day the pain was worse, now localized to the epigastrium and left upper quadrant. There were no nausea, vomiting or bowel changes and no further change in the neck swelling. There was no history of trauma. It is of interest to note a previous episode of left supraclavicular swelling which had occurred four months before, lasting several days with questionable associated mild abdominal pain.

Physical examination revealed a well-developed, healthy man complaining of severe abdominal pain. The temperature was 100° F.; pulse, 90; and respirations, 24. A soft, compressible nontender swelling was noted in the left supraclavicular area. The left lung base was dull, with diminished to absent breath sounds and no râles. Diaphragmatic excursions were poor. The heart was normal, and the blood pressure was 120/80. The abdominal wall was spastic and extremely tender in the epigastrium and left upper quadrant. No masses or borborygmi were noted. The rest of the physical examination, including a rectal examination, was negative.

The impression on admission was that the lesion might be a penetrating peptic ulcer, or possibly acute pancreatitis. Spontaneous pneumothorax was suggested but ruled out by the examination. Gastric aspiration removed 150 c.c. of clear fluid, with free acidity of 10 per cent and total acidity of 55 per cent.

Laboratory studies showed a hemoglobin of 78 per cent, subsequently rising to 100 per cent, and a white blood count of 13,500, with 72 per cent polymorphonuclear leucocytes. The urine was negative and the stools were guaiac negative on several occasions.

Conservative treatment was instituted, and a flat plate of the abdomen taken shortly after admission showed several loops of bowel in the left upper part of the abdomen to be distended with gas. In addition, a pleural effusion was demonstrated at the left lung base. An abdominal puncture was then done in the left upper quadrant, with removal of a droplet of fluid revealing a few polynuclear cells but no bacteria in spreads. Then chest aspiration was done through the ninth interspace in the left posterior axillary line and from 10 to 15 c.c. of a white milky fluid were easily removed. It had no odor, and on smear showed a moderate number of cells, mostly lymphocytes in a fibrin network on a background of tiny globules. Sudan III failed to stain these particles. Analysis of similar fluid removed subsequently from the neck revealed a total lipid content of 1.4 per cent, demonstrating the presence of chyle.

Because of the thoracic findings, the patient was transferred to our service.* He was observed and showed rapid subsidence of the symptoms until only the cervical swelling remained. The latter was aspirated and chylous fluid was withdrawn. It again failed to stain with sudan III. Culture revealed no growth. At this point one of us (H. N.) postulated the presence of a cervicomediastinal lymphangioma extending into the posterior mediastinum, and the subsequent procedures were carried out in attempting to establish this diagnosis.

Aspiration of the left chest no longer revealed fluid. Bronchoscopy was negative. The supraclavicular swelling had subsided, but despite this the area was reaspirated and 10 c.c. of the same milky fluid were again removed. (The analysis of this fluid showed 1.4 per cent total lipids and 52 mg. per cent cholesterol.) With the needle in place, 10 c.c. of iodized oil were injected with the hope of outlining the postulated structure. Roentgenograms taken over a period of two weeks showed migration of the iodized oil upward in the root of the neck, laterally toward the left axilla, and medially into the mediastinum. This movement of the iodized oil suggested its presence in preformed tissue spaces.

The patient now was completely asymptomatic and afebrile, but a definite diagnosis appeared to be warranted from the viewpoint of possible recurrence, possible therapy and also prognosis. There was no suggestion of underlying neoplastic disease and stools were guaiac negative on several occasions. It was therefore decided to explore the left supraclavicular region. This was done Feb. 28, 1944. No chylous fluid was encountered, but deep behind the clavicle a diffuse fatty mass, slightly whiter than normal and with occasional vesicles interspersed, was encountered and partially removed for study. The subclavian vein and thoracic duct were not exposed.

The microscopic examination† revealed areas of cystic dilatation with surrounding areas of lymphoid tissue as seen in cystic lymphangioma.

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*We are indebted to Dr. Ralph Colp for the privilege of further study and treatment of this patient, who was originally admitted to his service.

†By Dr. S. Otani and Dr. P. Klemperer.

A third case of an 8-year-old boy reported by Huber and Silver[29] is also of interest. There developed a pronounced nasal discharge, malaise and fever, followed in one week by vomiting and anorexia, and four days later by abdominal swelling and scrotal edema. He had ascites with edema of the back, thighs and scrotum. Several paracenteses removed milky fluid which contained fat on chemical analysis. Exploratory laparotomy was finally done because of rapid reaccumulation of the ascites, and the intestinal lacteals were found generally dilated throughout the gastrointestinal tract, along with enlarged, spongy glands in the mesentery. To facilitate drainage of peritoneal fluid, several large strands of silk were thrust through the parietal peritoneum into the soft tissues of both thighs and postoperatively the ascites did not recur.

In summary of these three cases, a common sequence of infection, followed in from one to two weeks by an effusion of chylous fluid in the thorax or abdomen, is seen, and at the time of report, two patients had recovered, with the third improving. No definite etiology could be found in those with chest involvement, while at laparotomy in the last patient, generalized obstruction and dilatation were noted in the abdominal lymphatics and could have represented the result of an old inflammatory process causing obstruction. Therefore we might postulate a congenital lymphangiomatous structure somewhere along the course of the thoracic duct in all three patients.

The occurrence of lymphangiomas in the neck is well known as exemplified by hygroma cysticum colli congenitum. But these are not always confined to the neck. They have been found to extend through muscle planes of the chest wall, up toward the jaw, and down behind the sternum into the anterior mediastinum. Dowd[28] reported two cases of lymphangioma extending into the mediastinum (1913), Singleton[24] added two more, Watson and McCarthy[21] mentioned their occurrence, and, most recently, Arnheim[22] reported two extensive cervicomediastinal lymphangiomas treated by surgery and injection of morrhuate. All of these cases were characterized by swelling with or without pressure symptoms.

SUMMARY

1. There is reported a unique case of cervicomediastinal lymphangioma, presumably arising from the thoracic duct or some of its radicles, which ruptured, causing a left chylothorax. The problem of diagnosis is discussed.

2. From a study of the case and a survey of the literature, a similar pathology and sequence of events in several previously reported cases of chylothorax of unknown etiology are postulated.

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A MUSCULOPLASTIC INCISION FOR POSTERIOR THORACOPLASTY

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The usual incision for posterior paravertebral thoracoplasty is carried straight down through the skin and underlying muscles to the ribs, making a J-shaped flap which is reflected forward. This method of making the incision has the following disadvantages:

1. It cuts through the thick muscle mass formed by the trapezius and rhomboids in the vertical part of the incision (Fig. 3) and through the latissimus dorsi and part of the serratus magnus in the lower part. Such willful section of big muscles offends the surgical conscience.

2. The hemorrhage, even under local anesthesia, is often considerable, is always undesirable, and may be so large in amount as to constitute a definite added risk in a feeble patient. The posterior scapular artery, as it runs down from above on the deep aspect of the rhomboids, is variable in position and may easily be divided.

3. That part of the muscles which lies between the incision and the spine must have its nerve supply cut, and almost certainly becomes atrophied and fibrous.

4. Much of this medial strip of muscle is indifferent material to hold a suture; the rhomboid layer is certainly of poor quality after division, and from the level of the fourth spinous process upward, the trapezius is aponeurotic and its fibers run largely transversely so that sutures obtain a poor grip. This medial strip of the aponeurotic portion of the trapezius probably has a poor blood supply and may easily undergo ischemic necrosis (Fig. 4).

5. It is particularly in this highest part of the incision that the wound is likely to give way when the stitches are removed. If this happens, it will usually be seen that the muscular layer had given way earlier, leaving the skin to take the whole strain.

6. If the wound does open, the direct superimposition of the skin and the muscle suture lines means that the extrafascial space is opened.

7. Although proper attention to the movements of the shoulder and scapula should result in no appreciable disability, it is difficult to believe that the muscles have not suffered permanent damage from the long incision.

8. The incision through the muscles is repeated at the second stage with consequent infliction of further injury.

For the last year I have been using a simple musculoplastic modification of this incision which seems to avoid most of these disadvantages. It is a simple and logical step and has proved very satisfactory in practice.

DESCRIPTION OF INCISION

The usual J-shaped incision is made down to, but not through, the muscles of the back. The vertical limb should be placed from two to two and one-half

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fingerbreadths from the midline. The medial flap is now undercut as far as the midline for the whole length of the vertical limb; the undercutting is continued into the first three to four inches of that part of the incision which curves forward, typically to just beyond the anterior border of the trapezius muscle. This undercutting is carried downward and medially for some two inches to expose the lowest part of the trapezius to within two inches of its lowest origin, where it is little more than one and one-half inches wide (Fig. 1). The most anterior part of the incision need not be undercut, but its lower edge should be retracted before the latissimus dorsi is divided. The incision in the latissimus dorsi is carried back to the lowest exposed portion of the trapezius, and this muscle is then cut through transversely to the midline. A finger is next inserted beneath the trapezius, immediately next to the origin from the spinous processes, care being taken to separate the muscle from the aponeurosis covering the erector spinae mass, to which it may be moderately adherent. With scissors or a knife, the trapezius is cut as near as possible to the spinous processes and interspinous ligaments (Fig. 2). With the finger separating the muscle in front of it, the division is carried right up to the top of the wound. The rhomboid muscles are, of course, raised with the trapezius when they are reached, and the serratus posterior superior may also be included, or left on the ribs, if desired. In the lowest part of the incision, the muscular fibers of the trapezius come so near to the midline that there is only a very narrow strip, barely 0.5 cm. wide, of aponeurotic tissue to be divided (Fig. 3). Higher up, this aponeurotic origin

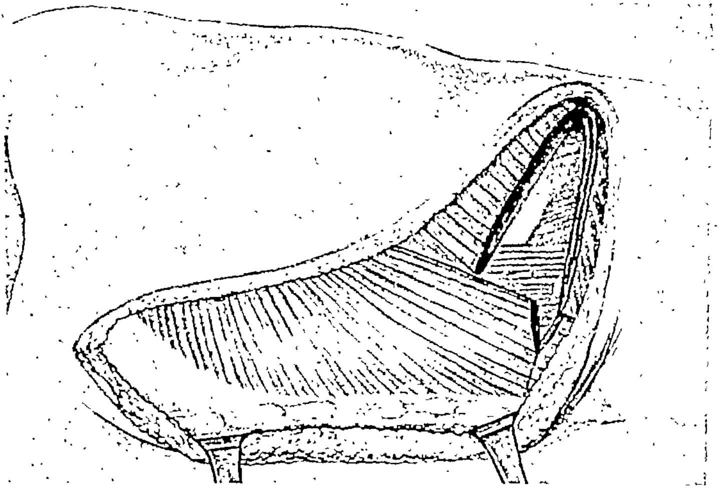

Fig. 1.—The vertical limb of the incision is shown undercut and retracted to expose the trapezius as far as its origin from the spine and spinous ligaments. Note how low the trapezius is divided.

TREATMENT OF SYPHILITIC ANEURYSMS BY CELLOPHANE WRAPPING

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THE surgical treatment of aneurysms still is an open field despite the various methods mentioned in the medical literature and even despite the modern surgical techniques available at the present time. According to Pearse,[1] the surgical attempt at producing progressive obliteration of the lumen of the great vessels appeared in the nineteenth century. Even before Moore in 1864 tried the wiring technique, small particles of metal were inserted into the aneurysm sac to encourage thrombosis. It soon became apparent that small, loose foreign bodies introduced into the aneurysm sac frequently escaped into the circulation, causing immediate death. The wiring of aneurysms then became popular and is still occasionally used. This consists of inserting from 30 to 200 feet of fine silver or copper wire into the aneurysm sac. A galvanic current was sometimes passed through the wire to encourage thrombus formation.

Dr. J. M. T. Finney, of Johns Hopkins Hospital, according to his personal communication to Emmert,[2] wired thirty-five aneurysms, with definite improvement reported in 50 per cent and no change in the other 50 per cent. It is interesting to note that while Finney reported that 50 per cent of the patients treated by wiring showed definite improvement, Babcock[3] mentioned statistics on thirty-nine patients treated by the same process at the Johns Hopkins Hospital, nearly 50 per cent of whom died in from one day to two months after wiring, and none of whom were known to be living at the time of Reed's report in 1926. Wheeler[4] reported on three patients operated upon in 1910, 1917, and 1928, in whom a whisp or a cage of Colt's wire was used; two patients survived four and eight years and one died three months after the operation from leakage of blood around the point of the wire into the aneurysm wall. This represents one of the most common and fatal complications following the wiring procedure. Even at the height of its popularity, the effectiveness of the wiring technique was recognized as limited to the saccular type of aneurysm, which is much less frequent than the fusiform type.

The anastomosis between the common carotid artery and the internal jugular vein is a method of treatment of aneurysms of the aorta advised by Babcock[3] but never very widely adopted. His procedure provides a by-pass which permits the blood of the aorta to escape to the lower negative pressure system of the vena cava.

A number of methods and devices have been advocated for producing occlusion of large blood vessels, with unsatisfactory results in many instances. In addition to the ineffectual results and fatal complications accompanying the

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use of many of these methods, almost all of them, with one or two exceptions, are too traumatic for application to a dilated, thin-walled syphilitic aneurysm which is about to rupture spontaneously. These methods of occlusion may be classified under three headings as follows: (1) insertion of foreign bodies into the lumen of the vessels to produce thrombosis; (2) external compression of the vessel; (3) injuries to the wall of the vessel, resulting in fibrosis and constriction of the lumen.

The results of insertion of foreign bodies into the lumen of the vessels have already been described as applied to the wiring of aneurysms. The method is applicable only to saccular aneurysms in which a current of blood is sufficiently sluggish to permit the development of thrombosis. It also has an appreciable mortality from leakage about the point of insertion of the wire.

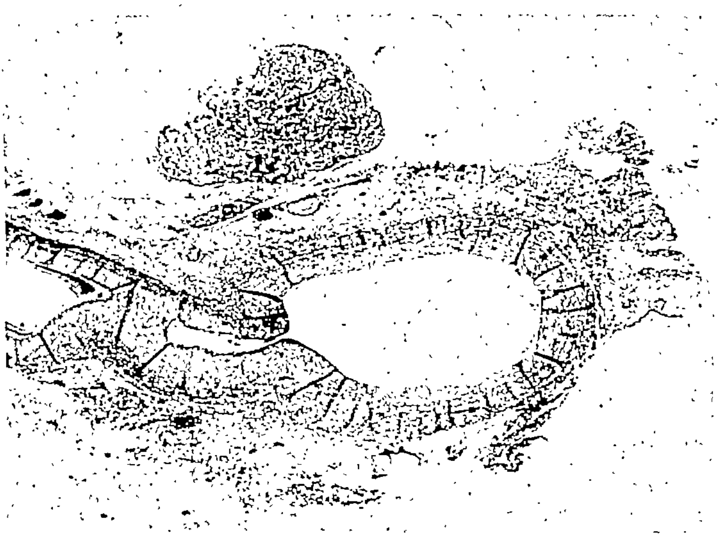

Fig. 1.—Cross section of aorta of dog wrapped for two weeks with physiologically inert 100 P.V. 000 type of cellophane. This section represents an almost normal aorta since almost no fibrous tissue reaction is present.

External compression of the wall of the vessel has been arranged in various forms to provide progressively increasing pressure by means of a spring device or an adjustable clamp regulated from outside the skin. Examples of this method are provided by the rubber tube of Matas, the Neff or Halsted clamp, the fascial strip and the Matas band. The disadvantages of this method are twofold: first, the pressure atrophy and consequent disruption of the vessel and, second, the tendency for infection to enter through the sinus tract.[5]

Most of the methods advocated for causing fibrosis and constriction by injury to the wall of the vessel are entirely too traumatic for consideration in

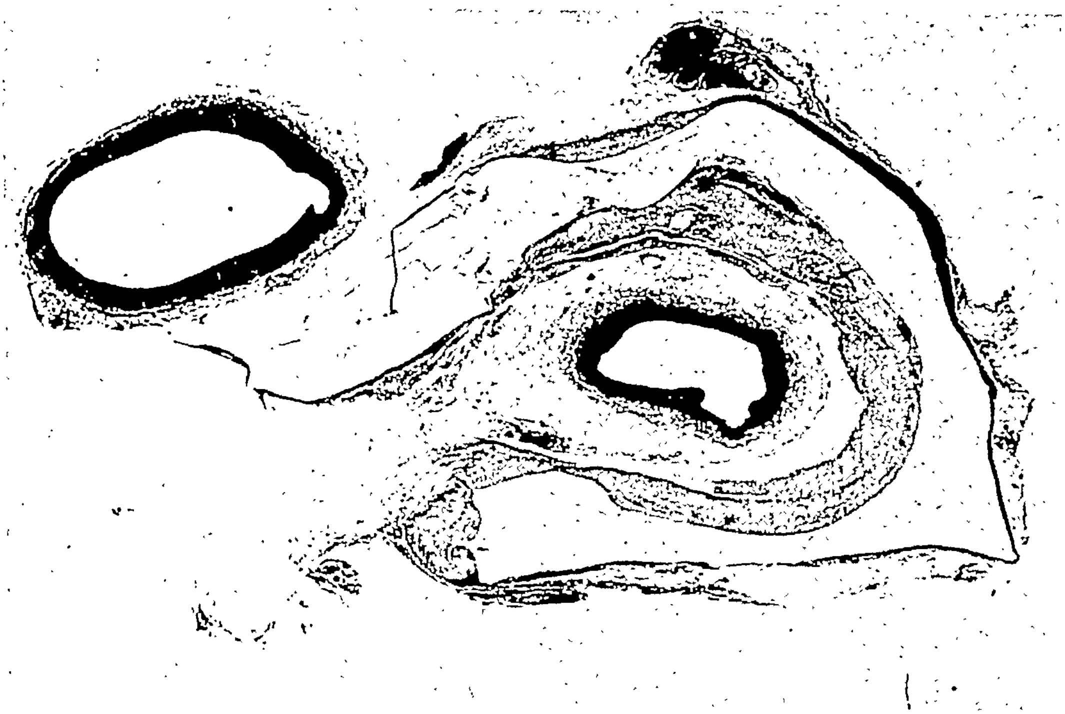

Fig. 5.—Two cross sections of aorta, lower right one of which was wrapped in polythene cellophane for two and one-half months. Note marked thickening of wall and decreased diameter of lumen in comparison with untreated control area represen ed in left upper corner.

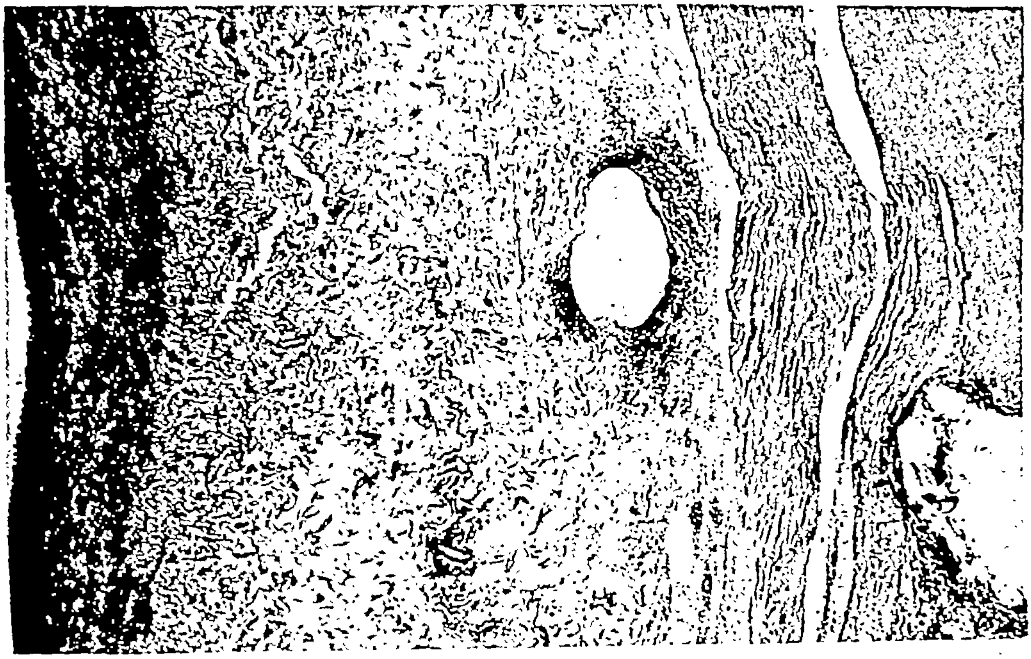

Fig. 6.—A section of thickened wall of aorta shown in Fig. 5 under higher magnification, illustrating fibrous nature of the reaction.

again two weeks later. A marked diminution of the murmur was noted two months later and complete disappearance, two and one-half months after its recurrence. No further recurrence of the murmur was noted in the one and one-half years during which the patient was followed. Similar reports are occurring more and more frequently in the medical literature. However, some dissatisfaction has been noted among some other surgeons who have used cellophane in attempting to obliterate the vessel lumen in cases of patent ductus arteriosus.

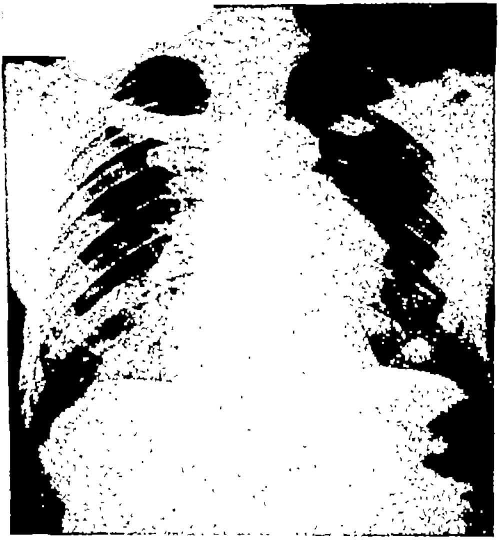

Fig. 7.—Roentgenogram of chest, posteroanterior view, illustrating fusiform aneurysm of descending aorta which was wrapped with polythene cellophane.

In direct contradistinction to the work of Page, Pearse, and Harrison and associates are the results of another group who considered cellophane as a physiologically inert substance suitable for application and insertion into areas of the body in which a foreign body reaction and adhesions would be very undesirable. Donati[11] was the first to demonstrate this inert property of cellophane in 1936. Regular commercial cellophane, .02 mm. in thickness, was used to prevent adhesions between the brain and skull, with evidence of no greater irritation than that caused by other materials. The cellophane was found close to the brain after thirty-five days with no irritative reaction surrounding it. Later, McKeever[12] used cellophane in the treatment of arthritis. He used type 300 P.U.T. 71 and concluded, as did Donati, that no reaction is caused by cellophane for at least two months after operation. This impression was confirmed by microscopic studies of a section of tissue taken from the vicinity of the cellophane in the injured knee.

Wheeldon[13] performed approximately fifty operations of arthroplasty, using 300 moisture-proof cellophane as the lining of joints. Again he agreed with McKeever, stating, "In this series of cases there has been no irritation of tissues by cellophane in any way." He had also used the material as a nonabsorbable suture and believed that irritation did not occur immediately after operation, or at any subsequent time.

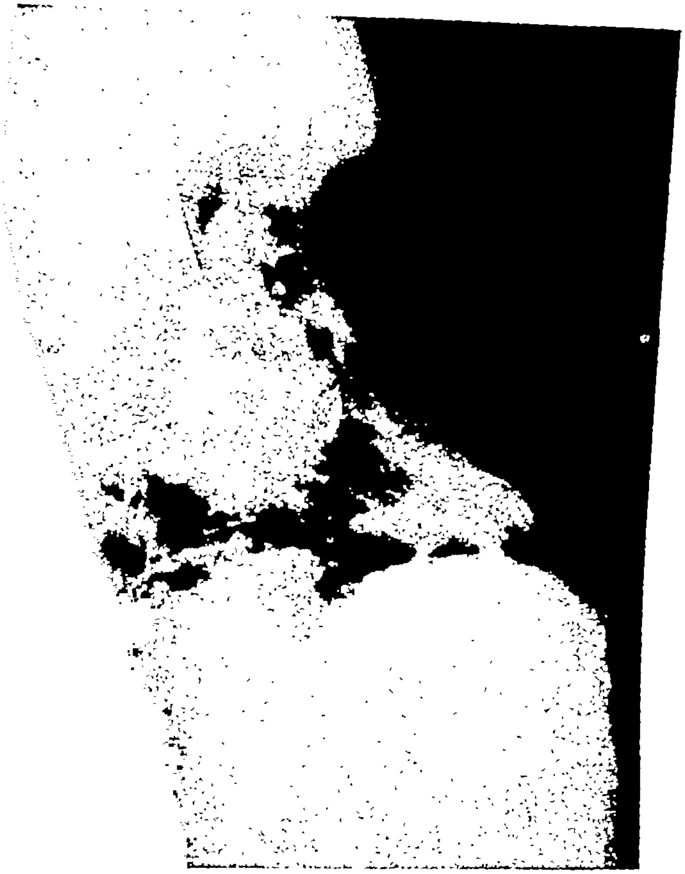

Fig. 8.—Roentgenogram, left lateral view, of chest shown in Fig. 7.

These various physiologic reactions of cellophane exhibited diametrically opposite properties. On one hand, cellophane represented an inert substance producing practically no foreign body reaction when left in the tissue and, on the other hand, inducing a violent fibrous tissue reaction resulting in fibrosis and contraction. This led to the conclusion that different varieties of cellophane produced entirely different physiologic reactions, depending upon the chemical structure of the material. Before any satisfactory clinical application could be made of the properties of cellophane to produce a constrictive fibrosis, a distinction was needed between the physiologically inactive and reactive types of cellophane. For that reason, a series of animal experiments was conducted on dogs, in which five different basic types of cellophane were used.

An investigation of different types of transparent films* revealed that only a few chemically different basic types are used. The large number of varieties is produced by combinations of these basic types with a wide variety of coatings to satisfy the packaging or other functional requirements of various industries. These coatings provide such quality characteristics as resistance to moisture vapor transmission in various degrees, heat sealing, packaging machine running qualities, etc. Some of these coatings resist the action of free water, whereas others do not. For these reasons, basic types of cellophane, or other films, have been used in most of these experiments with the results presented in Table I.

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*Supplied through the courtesy of the E. I. du Pont de Nemours and Company, Cellophane Division, Wilmington, Del.

TABLE I

TYPE OF FILM	CHARACTERISTICS	PHYSIOLOGIC REACTION
Cellophane 300 P.U.T. 71 300 P.T. 62	Cellulose hydrate; nonmoisture proof; insoluble in water and dilute acids	Little reaction
P.V. film 100B P.V. 000	Polyvinyl alcohol; water soluble	Little reaction; technical difficulties due to solubility of film
Polythene 1.5 mil	Insoluble in water and dilute acids	Marked fibrous reaction
Cellulose acetate 250 C.A. 40	Highly purified cellulose acetate film; insoluble in water and dilute acids	Little reaction

EXPERIMENTAL TECHNIQUE ON DOGS

Under nembutal anesthesia, a long, left pararectus incision was made from the costal margin downward. The abdominal aorta was exposed and freed throughout its entire length, preserving its major branches. Strips of double thickness cellophane of different varieties, each about one-half inch in width, were then passed around different sections of the aorta. Only two or three different strips were used on any one animal to provide a wide space between the different varieties of cellophane and to avoid overlapping of reactions. These different types of cellophane were carefully diagrammed as to location on each animal to assure accurate interpretation of the reaction at autopsy. A major branch of the abdominal aorta was used to separate the different strips to prevent their slipping together. The two ends of each strip were sutured together with fine silk to hold the ring of cellophane loosely around the aorta. The peritoneum was then closed loosely over the aorta and retroperitoneal space and the abdominal wall was closed.

Comments.—The abdominal aorta was used in preference to the thoracic aorta to eliminate the complications involved in providing positive intratracheal pressure to avoid pulmonary collapse. An unsuccessful attempt was made to produce sclerosis by painting the aorta with rose aniline dye similar to that used in the manufacture of indelible pencils, which is known to have produced a marked fibrotic reaction. This proved fatal due to the production of extensive necrosis of the liver and spleen. The period of observation in two of the animals was shortened by the development of an infection in one, apparently due to contamination at operation, and by distemper in another.

Results.—A very marked fibrous tissue reaction surrounding the section of aorta in close proximity to the polythene variety of cellophane was noted in

Wheeldon[13] performed approximately fifty operations of arthroplasty, using 300 moisture-proof cellophane as the lining of joints. Again he agreed with McKeever, stating, "In this series of cases there has been no irritation of tissues by cellophane in any way." He had also used the material as a nonabsorbable suture and believed that irritation did not occur immediately after operation, or at any subsequent time.

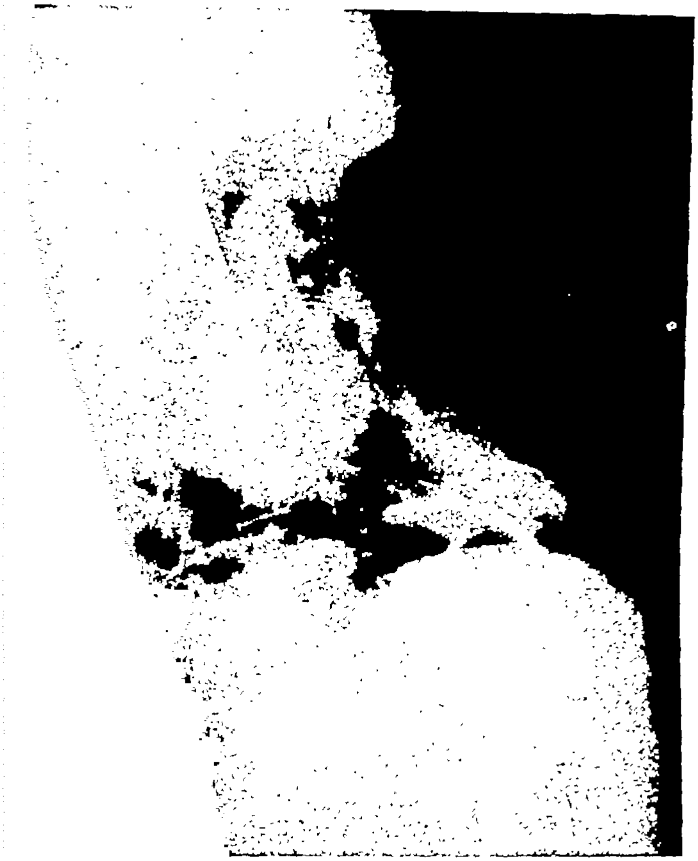

Fig. 8.—Roentgenogram, left lateral view, of chest shown in Fig. 7.

These various physiologic reactions of cellophane exhibited diametrically opposite properties. On one hand, cellophane represented an inert substance producing practically no foreign body reaction when left in the tissue and, on the other hand, inducing a violent fibrous tissue reaction resulting in fibrosis and contraction. This led to the conclusion that different varieties of cellophane produced entirely different physiologic reactions, depending upon the chemical structure of the material. Before any satisfactory clinical application could be made of the properties of cellophane to produce a constrictive fibrosis, a distinction was needed between the physiologically inactive and reactive types of cellophane. For that reason, a series of animal experiments was conducted on dogs, in which five different basic types of cellophane were used.

An investigation of different types of transparent films* revealed that only a few chemically different basic types are used. The large number of varieties at a thin, soft spot on its anterior wall where it rubbed against the posterior surface of the sternum with each pulsation and respiratory cycle. These other aneurysms extended to the right and caused marked distortion of the surrounding structures, particularly the superior vena cava and trachea. These structures required separation from the distorted aorta following incision of the mediastinal pleura. The sheet of cellophane was then slipped under the superior vena cava and the mediastinal pleura was drawn between the cellophane and vena cava to separate them and reduce the effect of the cellophane on the vena cava. Some difficulty was occasionally encountered in procuring sufficient mediastinal pleura to completely cover the cellophane. The most satisfactory way to avoid the contact of the surrounding structures with this extremely reactive polythene film seems to be to cover it with a layer of less reactive cellophane, such as 300 P.U.T. 71, 300 P.T. 62, or one of the moisture-resistant varieties. Further animal experiments are being carried out at the present time on refinements in technique, this representing a preliminary report.

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*Supplied through the courtesy of the E. I. du Pont de Nemours and Company, Cellophane Division, Wilmington, Del.

Polythene film was also used recently for the ligation of a recanulated ductus arteriosus in a 14-year-old colored girl with subacute bacterial endocarditis in whom the patent ductus had been ligated four years previously with cotton umbilical tape. Any attempt at transection of this poorly accessible Botallo's ductus, which was firmly bound down in scar tissue, would have been impossible and fatal. The use of a reactive type of cellophane in this case provided a highly satisfactory solution to a difficult problem. This would seem to present a satisfactory method of treating all patent ducti and a much less dangerous one than transecting such short and broad vessels between clamps. A certain amount of caution is necessary, however, to keep the cellophane away from the recurrent laryngeal nerve.

CONCLUSIONS

1. The use of polythene cellophane seems to offer a successful method of gradual fibrosis and obliteration of aneurysms and large vessels.

2. Experimental studies reported here have shown that in order to obtain the desired result, it is necessary to use a particular type of cellophane, since some varieties produce no fibrosis.

3. Polythene cellophane has been found useful not only in the treatment of aortic aneurysms, but also in the obliteration of a patent ductus arteriosus.

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CYSTIC DISEASE OF THE LUNGS AND ITS RELATIONSHIP TO BRONCHIECTATIC CAVITIES

A Study of Twenty-Two Cases

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and John R. McDonald, M.D.‡
Rochester, Minn.

A WIDE variety of names has been given to the condition commonly known as cystic disease of the lungs, These various terms indicate the different views which have been held as to the genesis of the cysts found in the lungs. Some of the more common names are pulmonary cysts, bronchogenic cysts, congenital cystic disease of the lungs, pneumocysts, honeycomb lung, solitary and balloon cysts of the lungs and pneumatoceles.

The term cystic disease of the lung, it seems to us, can be used for all abnormalities of pulmonary tissue in which cysts or cystlike structures are found, regardless of pathogenesis. It includes "cystic" bronchiectasis which refers to the rather large cystlike cavities sometimes produced by bronchiectasis but not to the ordinary cylindrical and saccular forms of bronchiectasis, mediastinal cysts of bronchiogenic type, nor to such specific entities as parasitic or hydatid cysts, tuberculous cavities, ordinary pulmonary abscesses with little or no attempt at epithelization of cavities or cavitary bronchogenic carcinoma. By broad definition, all pulmonary cysts or cystlike cavities may be considered forms of bronchiectasis; however, cystic disease of the lungs has commonly been considered a separate condition and is so regarded in this paper.

In this paper, too, the term congenital bronchiectasis has been avoided in connection with the cysts included under cystic disease of the lungs because it seems to us that its use has a tendency to confuse the issue. In our present state of knowledge, it is probably better to use the term congenital bronchiectasis only for what is believed to be the rare forms of bronchiectasis, which presumably are present from birth.

HISTORICAL ASPECTS AND REVIEW OF THE LITERATURE

Cystic disease of the lungs undoubtedly existed for many years without being recognized. As far as can be determined, no reports of pulmonary cysts, as such, appeared prior to the seventeenth century. The earliest report of cystic lungs was probably that by Thomas Bartholinus; a translation of his cases from the Leyden edition of Malpighius, in 1687, may be found in the article of recent years by Oughterson and Taffel.[1]

One of the best of the early twentieth century American descriptions of this condition was that by Koontz[2] in 1925, who was able to collect 108 cases

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from the world literature. He reviewed the findings at necropsy as reported in the literature and reported a case of his own. In the report of this case he included a detailed description of the pathologic changes.

Since 1925, the number of reports of cases of cystic disease of the lungs has increased greatly both in America and Great Britain. Among American writers, Wood and others,[3-7] Schenck[8, 9] and Maier[10, 11] recently have been outstanding contributors to knowledge of this condition. Among the British authors of recent times, Smith,[12] Sellors,[13] Willis[14] and Willis and Almeyda[15] have been prominent in the investigation of this condition.

Although cystic disease of the lungs is considered rare, Schenck,[8] in 1937, stated that a total of 381 cases of cystic disease of the lungs had been reported since the original description. Wood,[6] in August of the same year, added forty-eight cases in which the patients were seen at the Mayo Clinic. Within recent years, many excellent reports have appeared and the condition is being diagnosed with increased frequency and accuracy.

CLASSIFICATION OF CYSTIC DISEASE OF THE LUNGS

The following working classification is offered for cysts found in cystic disease of the lungs:

I. The true developmental or so-called congenital cysts of the lungs (some of the bronchogenic cysts or bronchoalveolar cysts)

II. Acquired cysts or cystlike cavities of the lungs
  A. "Cystic" bronchiectasis
  B. Other pulmonary cysts or cystlike cavities
    1. Pneumatoceles
    2. Abscess cysts
    3. All other pulmonary cysts, including some bronchogenic cysts (these pulmonary cysts predominantly result from infection, trauma [penetrating wounds of chest], noxious gases, hemorrhagic effusions and other contributory factors or combination of factors)
    4. Emphysematous blebs
    5. Emphysematous bullae

True developmental or so-called congenital cysts of the lungs are an important type of cyst. In addition to being located in the pulmonary parenchyma, they sometimes are found near or attached to the bifurcation of the trachea or to a main stem bronchus. The essential histologic feature of this type of cyst lies in the uniformity of the lining membrane which maintains the regular character of the bronchial epithelium (Fig. 1). In contrast to the regularity of arrangement of the bronchial epithelium is the disorderly arrangement and the erratic grouping of the supporting structures of the wall which is of the bronchial type. Cartilage, smooth muscle, elastic tissue and smooth glands are arranged irregularly. This arrangement is in marked contrast to the orderly arrangement of these structures associated with *simple dilatation* of a bronchus. An extreme variability in location and number of these features is almost characteristic of the developmental cyst. One or another of the stromal elements in these developmental cysts may be absent, or one of these elements may be prominent or even hyperplastic and, in a few instances, a picture almost like that of true neoplasia may result.

Of the acquired cysts, "cystic" bronchiectasis will be considered in detail in Table I, and the only other type which needs special mention is the pneumatocele. The pneumatocele has no epithelial lining and therefore may subside spontaneously. The distinction between "cystic" bronchiectasis and other acquired and congenital pulmonary cysts is difficult. The criteria given in Table I are of value in the histopathologic differentiation of "cystic" bronchiectasis from other cysts included under cystic disease of the lungs, especially the cysts of developmental or congenital origin.

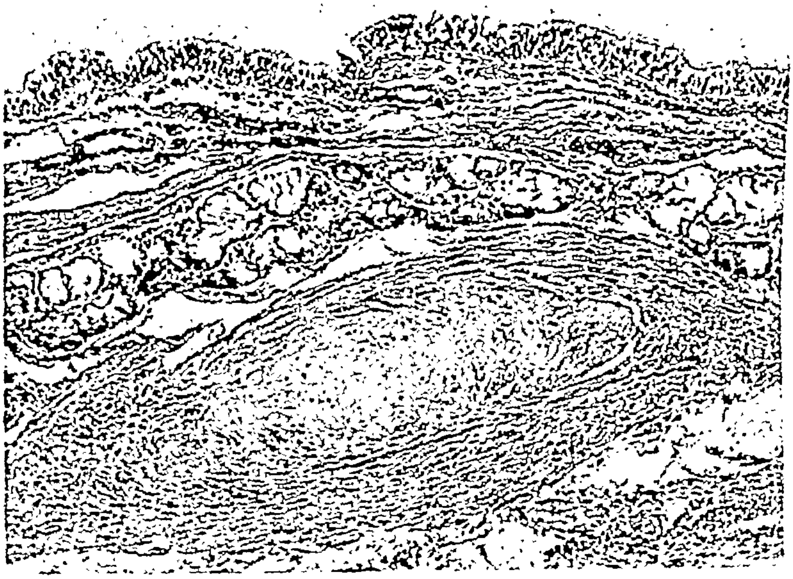

Fig. 1.—Portion of cyst wall shows the bronchial type of lining. The numerous mucous glands may be seen, as well as a portion of cartilage beneath the mucous glands (hematoxylin and eosin ×85).

STUDY OF TWENTY-TWO CASES

Twenty-two surgically treated patients with cystic disease of the lungs from whom tissue has been removed at the Mayo Clinic were selected for this study. No cysts of the mediastinum were included in this study although developmental or so-called congenital cysts of bronchial type do occur in the mediastinum. Each case was reviewed from the clinical standpoint and an analysis was made of the outstanding features. A detailed study also was made of the histopathologic features. Blocks were cut from the walls of each cyst and sections were prepared for microscopic study with hematoxylin and eosin stains in all cases; special stains, such as van Gieson's and mucicarmine, were used in selected cases. The character of the cyst lining was noted particularly. In addition,

TABLE I. COMPARISON OF PATHOLOGIC FEATURES OF "CYSTIC" BRONCHIECTASIS AND ALL OTHER TYPES OF CYSTIC DISEASE OF THE LUNG

	"CYSTIC" BRONCHIECTASIS	CYSTIC DISEASE OF THE LUNGS INCLUDING CONGENITAL OR DEVELOPMENTAL CYSTS
Size of cysts	Usually small, measuring from 2.0 to 3.5 or 4 cm. in diameter, but *may be larger*	Usually measures more than from 3 to 4 cm. in diameter and may reach huge dimensions
Location of cysts	Most frequently but not always in basilar lobes	Any place in lung or even in aberrant or accessory pulmonary tissue within thorax
Type of bronchial communication	Bronchi always involved and communicating with process; always enter cystic cavity directly	Bronchi may or may not enter cyst directly; bronchial communication may be small and imperfect but is usually present (rarely absent) and may be found on careful gross or microscopic examination (sometimes serial sections are needed to show it)
Usual relation of cysts to bronchi	Cystlike cavities are part of bronchiectatic process and are concentric to involved bronchi	Cysts are not part of a bronchiectatic process but bronchiectasis may develop secondarily; cysts are placed eccentrically in relation to bronchi
State of communicating or associated bronchi	Communicating bronchi always show dilatation and thickening of wall	Communicating bronchi may be normal, dilated or contracted
Condition of pulmonary parenchyma surrounding cysts	Usually considerable chronic pneumonitis with or without atelectasis	Not infrequently normal or minimal inflammatory process but occasionally chronic pneumonitis; atelectasis is often present about cyst
Microscopic findings in cyst cavities	Marked inflammatory process with irregular roughened lining; lining epithelium may be lost or ulcerated but there is a remarkable retention of epithelial lining; stromal elements show a regular arrangement or pattern	Lining epithelium is usually of bronchial type (stratified columnar epithelium with cilia present); in majority, bronchial elements in stroma are arranged *erratically* and these frequently show hyperplasia (sometimes to a considerable degree); some forms of cyst may have no epithelial lining and wall may be thick, fibrous and very inflammatory

the presence with the relative amount or absence of the following features was given consideration: inflammatory reaction, cartilage, mucous glands, lymphoid tissue, smooth muscle, elastic or fibrous connective tissue, vascularity, pigment, giant cells and the alveolar tissue.

The cystic disease of the lungs in these twenty-two patients could be classified into three groups as follows: developmental or congenital cysts in twelve, acquired cysts (including "cystic" bronchiectasis) in six and indeterminate cysts in four.

The final classification was arrived at by giving precedence to that feature of the case which seemed to be predominant. The largest group of cases was classed among the so-called congenital or developmental cysts. All of the cysts of this type had a definite bronchial type of lining. In the four patients in whom the cyst was of an indeterminate type, the lining was of an indeterminate type or simply fibrous in character. *Also the elements of the stroma* within the cyst wall did not suggest bronchogenic origin so that the condition in the four patients was classified in this way by necessity.

A summary of some of the important pathologic features in the twenty-two cases of pulmonary cysts may be seen by referring to Table II. Most of the cysts had a bronchial communication. Infection was present within the cyst in all

TABLE II. SOME OF THE FEATURES IN TWENTY-TWO SURGICAL CASES OF CYSTIC DISEASE OF THE LUNGS

CASE	AGE (YR.)	LOCATION OF CYST WITHIN LUNG	EXTENT OF INVOLVEMENT	BRONCHIAL COMMUNICATION	CONTENTS OF CYST	LINING OF CYST	TENTATIVE CLASSIFICATION OF TYPE OF CYST
1	38	Azygos lobe	From one-half to two-thirds of right upper hemithorax	Yes	Pus	Bronchial type	Developmental
2*	38	Right upper lobe	Upper one-third of right hemithorax	No	Thick, brown fluid	Bronchial type	Developmental
3	21	Left lower lobe	Limited to left lower lobe	Yes	Mucopus	Bronchial type	Developmental and acquired†
4	34	Left hemithorax	Practically all of left hemithorax	No	Chocolate-colored, thick material	Flat-celled indeterminate type	Indeterminate
5	12	Right upper and middle lobes	Right upper and middle lobes	No	Mucopus	Bronchial type	Developmental
6	26	Right lower lobe	Portion of right lower lobe	No	Mucopus	Bronchial type and squamous epithelium	Developmental and acquired†
7	47	Right lower lobe	Right lower lobe	No	Pus	Bronchial type and squamous epithelium	Developmental and acquired†
8	7	Left upper lobe	One-half of left upper lobe	Yes	Mucopus	Indeterminate type	Indeterminate
9*	13	Azygos lobe	Portion of right upper hemithorax	Yes	Mucoid material	Bronchial type	Developmental
10	25	Right upper and middle lobes	Right upper and middle lobes	Yes	Pus	Bronchial type	Developmental
11	26	Left upper lobe	Left upper lobe	Yes	Dark, clotted blood	Fibrous inflammatory wall	Indeterminate
12	30	Right lower lobe	Most of right lower lobe	No	Mucoid material	Bronchial type	Developmental
13*	27	Right upper and middle lobes	Right upper lobe and portion of middle lobes	Yes	Hemorrhagic mucopus	Bronchial type	Developmental
14	28	Upper one-third of left hemithorax	Upper one-third of left hemithorax	No	Mucoid material	Indeterminate type with flat cells	Indeterminate
15	18	Right lower lobe	Right lower lobe	No	Mucopus	Bronchial type	Developmental
16	43	Left lung	Most of left lung	Yes	Mucopus	Bronchial type and squamous epithelium	Developmental and acquired†
17	39	Right upper and middle lobes	Greater portion of right upper and middle lobes	Yes	Mucopus	Bronchial type	Acquired
18	36	Right lower lobe	Right lower and portion of right upper lobe	No	Mucopus	Squamous epithelium and bronchial type	Developmental
19	29	Right lower lobe	Portion of right lower lobe	Yes	Mucopus	Bronchial type	Acquired
20	45	Left lower lobe peripherally	Not significant	No	Thin, cloudy fluid	Fibrous inflammatory wall	Acquired
21	15	Left upper lobe	Left upper lobe	Yes	Mucopus	Bronchial type	Acquired
22	20	Right upper and lower lobes	Dorsal portion of right lower and small portion of right upper lobe	Yes	Mucopus	Fibrous inflammatory wall with ulceration of bronchial epithelium	Acquired

*No infection within the cyst.

†Developmental features predominant; therefore considered in text as developmental cysts.

but three cases. In none of the twenty-two patients in this series was there any evidence that bronchogenic carcinoma had its origin within the wall of one of these benign cysts.

One case has been selected as being fairly illustrative of all clinical and surgical features in the twenty-two cases of the pulmonary cysts and is presented in detail.

CASE 18 (Table II).—A woman, 36 years of age, complained of cough, productive of thick, purulent sputum, hemoptysis and frequent colds. These symptoms had been present for six years. Onset had followed pneumonia. The patient was in bed for eleven months at the time of the first attack and had had several pulmonary hemorrhages since the initial onset. Her disability because of symptoms previously mentioned and fatigue was considerable. Physical examination at the clinic revealed elevation of the right side of the diaphragm and decreased breath sounds over the base of the right lung. Multiple roentgenologic examinations revealed a tumor of the lower lobe of the right lung posteriorly, which apparently was connected to the mediastinum. The roentgenologist considered the possibility of a bronchogenic cyst. The sputum was repeatedly negative for tubercle bacilli. Bronchoscopy was performed, at which time it was noted that pus was coming from the apical branch of the bronchus of the right lower lobe. Operation was recommended because of the failure of conservative measures to give relief. A primary right posterolateral incision was made May 1, 1944, and a long segment of the sixth rib was resected. A large cyst was palpable in the upper portion of the lower lobe. The inflammatory reaction was marked around the cyst and extended into the adjacent portion of the upper lobe. An attempt was made to establish a

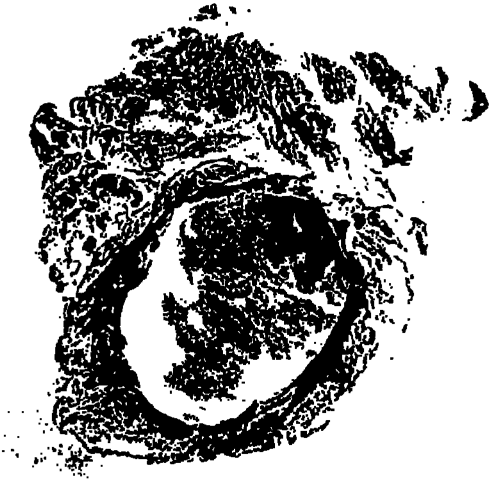

Fig. 2.—Large pulmonary cyst occupying the greater portion of the right lower lobe of the lung.

line of cleavage between the upper and lower lobes but, in doing so, the cyst was ruptured and considerable contamination resulted. The cyst was large, measuring about 8 or 9 cm. in diameter, and contained some foul debris. Dissection and individual ligation of the hilar structures were carried out and the right lower lobe was removed. Closed drainage was instituted. Bronchoscopy was performed at the end of the operation. The operative diagnosis was infected cyst of the right lung.

Gross examination of the tissue removed (Fig. 2) revealed a large cyst in the lobe of the right lung. The excised right lower lobe was largely occupied by the cyst, which measured 7 by 7 by 6 cm. and was located in line with the bronchus to the right lower lobe. On gross examination no communication was found between the bronchi and the cyst. The bronchial radicles were compressed by the cyst. The lining of the cyst was rather smooth and the surrounding pulmonary tissue was atelectatic. On microscopic examination the cyst was lined with pseudostratified columnar epithelium of the bronchial type, with a ciliated border in some places, but a large portion of the sections showed a squamous type of epithelial lining. Considerable inflammation was present in the wall of the cyst with ulceration of the lining. Cartilage and smooth muscle were noted in the wall of the cyst. Mucous glands were numerous and fibrous connective tissue was a prominent feature of the stroma within the wall. A small amount of lymphoid tissue was present. Small and large blood vessels were abundant throughout the wall of the cyst. Anthracotic pigment was noted within the pulmonary parenchyma surrounding the cyst. The parenchyma also showed chronic pneumonitis. After operation, secondary anemia and empyema developed. The empyema subsided after open operation by one of us (O. T. C.). Follow-up examination revealed that the surgical result was satisfactory.

COMMENT

From this study there appear to be many factors in the etiology and pathogenesis of cystic disease of the lungs which may be impossible to determine in a given case with any degree of certainty. Developmental factors, infection with its sequelae, mechanical and physiologic factors are all concerned as sources of origin of these pulmonary cysts. It appears unlikely that these cysts can be classified with assurance as being totally congenital (developmental) or totally acquired. For example, the so-called congenital cysts may be influenced profoundly by acquired factors and a cyst that is thought to be acquired may be influenced a great deal by the presence of a malformation or developmental anómaly. In our present state of knowledge, it does not seem possible to state whether a small, actual or potential cyst may exist at birth and may be detected for the first time later in life. Some developmental or congenital cysts have been reported as developing during life and, on comparison with previous roentgenograms, were not present earlier in life. The acquired nature, however, may have been only apparent and not actual. On the other hand, some pulmonary cysts do appear to be predominantly acquired. The large size of the pulmonary cyst, the eccentric position of the cyst in relation to communicating bronchi, normal communicating bronchi, a bronchial wall with erratic arrangements of stromal elements and hyperplasia of the latter suggest a developmental or congenital origin. The developmental or so-called congenital origin is more certain in cysts found in accessory or aberrant pulmonary tissue, or in mediastinal cysts with a bronchial type of wall.

Cystic disease of the lungs may be extremely variable in regard to the size, location and the resulting complications of the cysts. The pathologic picture at operation may be simple and uncomplicated, or it may be bizarre in the presence of hemorrhage and infection within the cyst and their resulting complications. The presence of pulmonary cysts and their complications may profoundly alter intrathoracic physiology. Accessory or aberrant lobes of the lungs may be associated with anomalous vessels and both are occasionally encountered in conjunction with cystic disease of the lungs. The bronchial communication present in cystic disease frequently results in the patient's coming to the attention of the surgeon.

Diagnosis.—The symptoms of cystic disease of the lungs are protean and are largely dependent on the size of the cyst, its location and the presence or absence of a bronchial communication. Hemoptysis or even severe pulmonary hemorrhage is not an uncommon symptom in cystic disease of the lungs. Hemoptysis and even severe hemorrhages were present in sixteen of the twenty-two patients in this series. A multitude of complications occurring with cystic disease of the lungs may simulate most any condition found in the chest. Pulmonary tuberculosis and bronchogenic carcinoma may coexist with cystic disease of the lungs. Pulmonary tuberculosis is a diagnosis which has frequently been made in these cases; the condition of a number of patients in our group had been so diagnosed and they had even spent long periods in sanatoria.

An accurate diagnosis of cystic disease of the lungs is largely dependent upon adequate roentgenographic examination of the thorax. It is felt that bronchoscopy is of definite value, especially for the patient who is to be treated surgically, to rule out any possibility of other bronchial lesions. Occasionally, an accurate diagnosis can be arrived at only by exploratory thoracotomy.

In the differential diagnosis, consideration must be given to almost all intrathoracic neoplasms, pulmonary abscess, localized empyema, tuberculous cavities, hydatid cysts and diaphragmatic hernias. Some eminent thoracic surgeons have admittedly treated what they thought originally was localized empyema only to discover, after failure of the cavity to become obliterated, that they were dealing with true pulmonary cysts.

Surgical Management.—Sellors[13] has pointed out how poorly the condition has been treated in the past and remarked that "the treatment of cystic disease of the lungs does not make satisfactory reading, and the number of cases that have been aspirated or drained without further attempt at cure shows how little the condition is understood."

The ideal treatment for cystic disease of the lungs is complete operative removal of the cystic portion of the lung with conservation of as much normal functioning lung as possible. Local excision is only occasionally feasible, as in a case in which the cyst may be attached by a narrow pedicle or loosely attached to the parenchyma of the lung. Most frequently it is far better to excise the entire cystic portion of lung if the distribution makes this possible and provided all other indications for such excision have been met. Drainage of the cysts and other supplementary operations, such as collapse procedures, usually are not satisfactory. Age appears to be no contraindication to surgical treatment. Operative treatment is being performed in the majority of these patients with safety, but, in some, the presence of complicating factors makes operation hazardous.

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THE MANAGEMENT OF THORACIC AND THORACOABDOMINAL WOUNDS IN THE FORWARD AREAS IN THE SICILIAN AND ITALIAN CAMPAIGNS

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THE purpose of this paper is to report the immediate results and analyze the methods employed in the management of thoracic and thoracoabdominal war wounds in the forward areas. This work was done by a surgical team of the Second Auxiliary Surgical Group during a twenty-month period, commencing with the invasion of Sicily. The employment and functions of surgical teams of an auxiliary surgical group have been described by Forsee.[1] Two hundred patients suffering intrathoracic and thoracoabdominal wounds treated in a first priority surgical hospital (a platoon of a field hospital located in physical conjunction with a division clearing station and receiving severely injured casualties who could not be transported farther to the rear, without surgery, than the rear of the division boundary) or an evacuation hospital constitute the principal basis for this paper. Sixty-four cases will be reported, in which wounds were limited to the chest wall, and the relationship of intrathoracic to spinal cord injuries will also be discussed. During the twenty months, the team functioned as a thoracic surgical team for one-half the period and as a general surgical team for the remainder. Our experience has led us to believe that intrathoracic injuries are more common than intra-abdominal wounds and that thoracoabdominal wounds constitute approximately one-quarter of all thoracic injuries with pleural involvement. The patients treated incurred the following types of wounds as their major injury: 84 (42.0 per cent) penetrating, 61 (30.5 per cent) perforating, and 55 (27.5 per cent) thoracoabdominal. The total mortality was 9.0 per cent (18 deaths). Excluding the thoracoabdominal wounds, the mortality for the 145 intrathoracic cases was 6.2 per cent (9 deaths).

The length of time during which the patients were retained under our immediate observation is of interest because, with early surgery possible, the majority of the nontransportable patients soon became transportable. Patients with thoracic wounds were retained in the hospital an average of seven days, the shortest four days and the longest fourteen days. The thoracoabdominal patients were kept an average of ten days, the shortest eight days and the longest nineteen days. The presence of concomitant major wounds was the cause of retaining patients longer than the average periods stated here. The policy of evacuating the patients to a hospital in the base section, designated as a chest center, as soon as they were safely transportable enabled the majority to arrive in good condition and placed them in the care of experienced thoracic surgeons for any further treatment required during the optimum period of their illness. Therefore, this report emphasizes only the immediate results, and the mortality rates presented stand uncorrected for development of subsequent complications and for the lethality of concomitant wounds. It is essential, however, to note

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that 42.5 per cent of the 200 patients incurred concomitant severe injuries which often were important factors in determining the urgency of operation as well as the extent and nature of the surgery to be performed. In a subsequent report of a final follow-up to be completed, the influence of concomitant wounds and complications will be discussed. No originality is claimed for the observations or methods described in this report and proper credit cannot be given because of lack of reference facilities.

TYPES OF MISSILES

Bullets have less tearing action than do high-explosive fragments and they have two characteristics that are of interest. They cause considerable contusion to the organs through which they pass, and frequently they travel greater distances than do fragments. In bullet wounds necessitating thoracotomy, the large amount of pulmonary hematoma surrounding the tract of the missile is considerably greater than one expects from either the size of the missile or the roentgenographic findings. This has been noted repeatedly in lower lobes in thoracoabdominal wounds. Whether this is due to the wobbling of the bullet or to the high compression wave accompanying it, we do not know. The tendency of a bullet to involve more than one celomic cavity is striking. An example is a case in which a bullet entered through the left deltoid region and was found in the right iliac fossa—the patient had been lying down with his left arm extended above his head at the time of injury. Another example is that in which a bullet entered through the left clavicle and was found near the greater trochanter of the left femur.

PREOPERATIVE TREATMENT

The purpose of preoperative treatment is to restore the disturbed cardiorespiratory mechanism to as close an approximation of the normal as possible. The principles in the restoration are those involved in any seriously wounded patient to which are added certain factors peculiar to intrathoracic wounds. These are airway, chest wall pain, intravenous fluids, hemopneumothorax, and progressive tension pneumothorax.

The airway is of primary importance and the relief of pain is often essential in obtaining an open airway. In the majority of patients there is blood in the tracheobronchial tree. The amount varies from mere blood-tinged sputum to sudden massive hemoptysis. Fortunately, the latter is rare.[*] Encouraging the patient to cough will often suffice to clear the tracheobronchial tree. Manually holding the lower costal margin will aid in coughing. Chest wall pain may inhibit coughing and limit the depth of respiration, resulting in an inefficient hyperpnea with inadequate ventilation. This is best treated not by morphine, but by intercostal nerve block. The block should include not only the segments involved by the wound but also two segments above and below to cover the area of pain overflow. If the wound is close to the vertebral column, injection of the intercostal nerves is *difficult, and a paravertebral block should be done.* The relief of pain will permit the patient to cough without difficulty. Clearing of the tracheobronchial tree will rapidly change the hyperpneic, cyanotic, hypotensive patient to one with deep unhurried respirations, good color, and rather prompt return of blood pressure to a more normal level. The resultant open airway, by relieving the hypoxia, will more readily raise the blood pressure than will any other procedure, except in the patients with continuing intrathoracic bleeding and progressive tension pneumothorax. Endotracheal catheter suction is imperative in clearing the airway of the unconscious patient and of the one whose cough is inadequate. In all cases of hypoxia and shock, oxygen should be given by nasal catheter. Oxygen administration should precede all preoperative measures and should be continued until the patient is ready for operation.

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[*] An example of massive hemoptysis is that of a soldier wounded by a high explosive fragment perforating the right upper part of the chest. Shortly after admission to a field hospital, the patient had a sudden massive hemorrhage which filled the entire trachea and primary branches. Bronchoscopy cleared the trachea for the moment, but hemorrhage from the orifice of the right upper lobe bronchus continued. The hemorrhage was controlled only by packing its orifice, and the packing was held in place by also packing the entire right main bronchus. This permitted a thoracotomy to be done with suture of the bronchus and bleeding lung. The patient was seen one month later and had a fully expanded lung.

The position of the patient and caution in administration of intravenous fluids are important. Vital capacity is greater and coughing is more effective in the sitting position. The ideal may be approached by placing the patient in a semisitting position. However, patients with hypotension and no cyanosis or dyspnea should be placed in a Trendelenburg position. In the administration of blood, it should be remembered that a patient with partial or complete pneumothorax may develop pulmonary edema by the sudden overloading of the pulmonary vascular bed. This is especially true in patients with hypoxia due to reduction in vital capacity by mechanical means such as hemopneumothorax. The reduction in the vascular bed is a sudden one and the patients does not have time to compensate. The vascular bed is not only reduced by the partial collapse of the lung but is further reduced by the extensive hematoma of the pulmonary parenchyma which may be present. Blood replacement therapy can be given judiciously by watching for moisture in the pulmonary parenchyma. Intravenous fluids should not be started on a patient who is having difficulty coughing in the presence of gross rhonchi. The airway can be cleared, pain reduced, and thoracentesis performed in a short time, and, with relief of hypoxia, the pulmonary vascular bed will not react unfavorably to intravenous fluids.

In our experience, hemopneumothorax was so commonly encountered that treatment of its immediate effects was often an urgent requirement. The total number of hemopneumothoraces in the 200 cases was 159 (79.5 per cent). Hemopneumothorax occurred unilaterally 149 times, and bilaterally 5 times. Hemothorax alone was found in 25 cases and pneumothorax alone in 10. Neither pneumo- nor hemothorax was discernible in 4 patients. Thoracentesis was performed on the majority. This procedure assists in relieving shock by the relief *of hypoxia consequent to the increased aerating parenchyma.* For this reason, as much blood and air as can be obtained is removed. The largest amount was 1,100 c.c., and the average was 250 c.c. Autotransfusion has been used frequently. Occasionally the blood clots, especially if the bleeding is rapid; therefore, it is citrated as it is withdrawn and strained through gauze before being given.

The possibility of the resumption of bleeding as a result of immediate thoracentesis is frequently discussed, but is more a theoretical problem than a practical one. The two sources of hemothorax are extrapulmonary (chest wall or mediastinal) and pulmonary hemorrhage. When resumption of bleeding does not occur it is probably due to the low pressure in the pulmonary artery and its branches. The presence of a hemothorax does not stop extrapulmonary bleeding. The objective of preoperative treatment is rapid resuscitation and thoracentesis helps accomplish it. Thus, theoretical contraindications need not be considered.

Thoracentesis was performed 100 times, and, in 73, pleural pressure readings were taken as the first step in the procedure. Pressures were recorded pre- and postoperatively and were correlated with the pre- and postoperative condition of the patient. The average positive pressure on expiration was 10 to 12 cm. of water. A few patients had positive readings on both expiration and inspiration. Following the removal of air and blood, the pressures more nearly approximated the normal. It was the exceptional patient, however, who at the end of a thoracentesis gave negative readings on *both* expiration and inspiration. There was not always a correlation between the degree of dyspnea and the primary pleural pressure. This was probably due to the variation in the stability of the mediastinum, with a corresponding individual variation in the transmission of transmediastinal pressures. Not all patients with positive pressures on inspiration and expiration were dyspneic, but the majority of the severely dyspneic ones presented positive pressure readings in the entire respiratory cycle.

It is of interest to note that true progressive tension pneumothorax was rare in this series. It soon became obvious, in the management of these cases, that those which required constant decompressing devices were quite rare. In only three instances was it necessary to install a decompressing catheter when the patient was first seen, and only two patients developed a true progressive tension pneumothorax postoperatively. The designation, progressive tension pneumothorax, was reserved for the patients requiring constant decompression devices. The mere fact that positive pleural readings were obtained at the first thoracentesis, in the majority of cases, makes it reasonable to classify them on the basis of treatment necessary because these patients required only intermittent thoracentesis postoperatively. We believe that most of the initial positive readings result from air from the injured lung entering the pleural cavity at the time of wounding, and the pressures may be increased from bleeding into the pleural cavity. The continued air leaks must nearly always stop soon after wounding, or remain constantly minimal, because in all cases the pulmonary involvement ranged from small penetrations or perforations to large lacerations and yet the rarity of progressive tension pneumothorax was rather striking. Perhaps the hematoma surrounding the area of the torn parenchyma creates sufficient nonaerating lung to limit the amount of air reaching the pleural cavity. The amount of pulmonary hematoma surrounding even a small tract is often considerable. Three-quarters of a lobe may be involved from the passage of a small fragment.

Immediately upon admission, the patient should be examined for sucking wounds. If one is present, it should be dressed with a large occlusive petrolatum dressing. It is good practice to dress all wounds of the chest in this manner, for a wound labeled nonsucking may begin to suck when the position of the arm or shoulder is changed.

Roentgenograms of the chest can be obtained as soon as the patient responds to resuscitation. Erect posteroanterior and lateral views are those of choice. However, it is not always possible to have the patient sit up. If not, one must be satisfied with a prone anteroposterior view. Fluoroscopy is of value in determining the amplitude of the cardiac pulsation in suspected tamponade. It is also valuable for the few patients who do not respond to resuscitation and for whom the additional movement necessary for roentgenography is deemed inadvisable. The position of the missile can be determined in order to aid in assuming what cavities have been invaded and what structures have been involved.

Thoracoabdominal wounds require the same preoperative care, and, in addition, a Levine tube is inserted, the stomach is aspirated, and the tube is left in place. During the operation, the tube keeps the stomach deflated and aids exposure, particularly when the left transdiaphragmatic approach is being used. The tube should be no smaller than a No. 18 French. The presence of blood in the gastric contents is of no aid in assuming involvement of the stomach, for the patient usually swallows the blood he does not cough up. In perforating wounds with one of the wounds in the region of the kidney or in penetrating wounds with the missile in the flank, the urine should be examined for the presence of blood.

In the patients who do not respond adequately to resuscitation measures and in whom the chest involvement is felt to be under control, the factors of continued intraperitoneal hemorrhage and/or continued intraperitoneal contamination from large bowel contents should be kept in mind. Gross contamination of the left pleural cavity by contents of the large bowel will result in as profound a depth of shock as is seen. These patients will respond, if at all, only after the source of the contamination has been removed. Early surgical intervention is necessary when the right lobe of the liver continues to bleed into the pleural cavity or when the spleen lies shattered in the left pleural cavity. One must be able, on the basis of the position of the wound of entrance and the position of the fragment in relation to the lack of response to shock therapy, to evaluate the probable situation and proceed with surgery without further delay in spite of the hypotension. The return of a more normal blood pressure after clamping the splenic vessels or packing the liver is most gratifying. But its return in fecal contamination of the peritoneum and/or pleura is not so prompt and may not occur if the contamination has been severe and of long duration. There is also an occasional case in which the airway becomes the primary problem because of excessive bleeding into the tracheobronchial tree. If bleeding continues after a thorough endotracheal catheter suction, further delay is unwise, and the patient is removed immediately to the operating tent for rapid induction and endotracheal intubation which permits a more complete tracheobronchial toilet to be done. In our experience this method of management of the latter group of patients has so improved their general condition that the necessary surgery could be accomplished without further delay.

PENETRATING AND PERFORATING WOUNDS

Treatment of penetrating and perforating wounds is, in the main, based on the presence or absence of continued intrathoracic hemorrhage. Thoracotomy is necessary to control those which are bleeding, whereas those not bleeding are treated according to the anatomic severity of the injury. The latter are treated by local débridement and closure of deep muscle layers. Sucking wounds require enlargement for proper closure. Wounds causing large chest wall defects will be discussed under Traumatic Thoracotomy.

The diagnosis of continuing intrathoracic hemorrhage is the most difficult diagnostic problem in thoracic surgery in forward installations. It has been pointed out that death from hemorrhage from the pulmonary parenchyma is unlikely. We saw four instances of bleeding of considerable magnitude from lacerations of the upper half of the upper lobes which required suturing. There was no similar degree of hemorrhage from lacerations of the same size in the remaining lobes. The reason may be that lacerations in this region involve vessels of greater size than comparable lacerations in other lobes. There remain the following sources of hemorrhage: intercostal (10 cases), internal mammary (2 cases), and mediastinal vessels (2 cases—aorta, hemiazygos, and accessory hemiazygos veins), and the heart (2 cases). These illustrate the fact that intrathoracic hemorrhage from sources other than intercostal vessels is relatively uncommon. Originally, we believed that intercostal vessels in the wounds should be visualized in the majority of cases. A record was kept of all intercostal arteries which were bleeding at the time of exposure and those which were transected or lacerated but not bleeding. In 200 cases, an intercostal artery was found bleeding only 10 times (5.0 per cent), whereas it was found transected or lacerated but not bleeding 33 times (16.5 per cent). All bleeding vessels were found in one of two areas: first, in the area from the origin of the vessel to the angle of the rib and, second, in an area extending approximately 6 cm. from the lateral border of the sternum. In view of these findings, it was felt that more conservative treatment could be practiced. In the latter part of the series, 35 penetrating and perforating wounds were locally débrided without investigating the continuity of the intercostal vessels. There were no deaths in this group. It is reasonable to assume that some of these patients had involved but nonbleeding intercostal vessels. This makes the proportion of nonbleeding to bleeding intercostal arteries more marked.

The knowledge of the percentage chance of the presence or absence of a bleeding intercostal artery is not of great help in the individual case. Each case must be carefully evaluated on the basis of individual findings. The presence of a large hemothorax is not an indication for an exploratory thoracotomy. Aspiration can be done and a check roentgenogram and reaspiration will permit the evaluation of the patient's status. This program works well during periods of relative quiet when only a few patients are admitted. During a "push," however, the surgeon is presented with a large number of patients, and his judgment will often be sorely tried. During the period of observation of patients with suspected bleeding, the traumatic thoracotomies and thoracoabdominal wounds can be treated. To attempt a diagnosis of continued intrathoracic bleeding solely on the basis of changes in pulse rate, blood pressure, and respiration will not infrequently lead to error, for some patients remain in relatively good condition with actively bleeding intercostal vessels. Judgment of the problem cases becomes more accurate in proportion to the number of cases seen. No deaths from bleeding vessels have occurred in this series. However, from the number of involved but nonbleeding intercostal arteries which we have uncovered, we feel that it is better to investigate a case if any doubt exists. One should keep in mind the possibility of the postoperative recurrence of intercostal hemorrhage in the patients whose intercostal vessels were not exposed. It did not occur in this series, but we have been informed of two fatalities from hemorrhage on the second and fourth postoperative days.

In wounds which were not sucking and were not the source of bleeding, débridement of the chest wall muscle was done avoiding, when possible, the opening into the pleural cavity. Endotracheal anesthesia was used, because occasionally a simple wound started sucking during débridement. After débridement, these wounds should have at least a two-layer closure of the chest wall muscle. Skin and subcutaneous tissue were left unsutured.

Sucking wounds necessitated an exposure adequate for a proper closure. With the wound in the intercostal space, the pleura had a defect best closed by including a deep layer of the chest wall muscle in the line of sutures which included the intercostal muscle. At least two more muscle layers were usually available for closure with interrupted silk sutures. In the anterior portion of the lower costal margin, imbrication of the single muscle layer of the chest wall was desirable in order that more than one muscle layer, besides the intercostal one, be used in closure. When the wound involved a rib, the fragments dangling into the pleural cavity were removed. If the intercostal nerve was readily found, it was crushed to relieve postoperative pain. The continuity of the intercostal vessels was checked at this time. The skin and subcutaneous tissue were left unsutured.

The problem of removing foreign bodies from the lung or pleural cavity should be considered in the light that in the majority of patients they are tolerated without endangering life. Removal of a foreign body is not of itself sufficient indication for thoracotomy in the forward areas. The exceptions to this generalization are the patients in whom the foreign bodies are in potentially dangerous positions, such as in the hilus of the lung or in the mediastinum in the region of the esophagus. However, during closure of a sucking wound, exploration for source of continued bleeding, or exploration to investigate diaphragmatic involvement, any readily available foreign body should be removed. The position of the foreign body was recorded in 75 of the 84 penetrating wounds. Forty-one (54.7 per cent) were found in the lung, 18 (24.0 per cent) in the pleural cavity, and 16 (21.3 per cent) in the chest wall; the last traversed the lung. In twelve patients, the foreign bodies were readily available in the lung during surgery for sucking wounds and were removed. In two instances, thoracotomy was done because of the presence of missiles. In both cases, they were very large and in the hilus of a lobe. Foreign bodies were removed from the pleural cavity in thirteen patients. In five, thoracotomy was sided, and 3 bilateral. In the last, a single missile involved both pleural cavities and both diaphragms. There were 9 deaths (16.4 per cent). This mortality is uncorrected for the factor of concomitant wounds. Intra-abdominal organs were involved as shown in Table I.

TABLE I. INVOLVEMENT OF INTRA-ABDOMINAL ORGANS

ORGAN	NUMBER	PER CENT
Liver	38	69.1
Spleen	12	21.8
Stomach	10	18.1
Large bowel	7	12.7
Kidney	6	10.9
Small bowel	3	5.4
Pancreas	2	3.6
Gall bladder	1	1.8
Common duct	1	1.8

The mortality is greater with left-sided wounds in which massive contamination of the pleural cavity and/or peritoneal cavity from contents of the large bowel occurs. Of the 19 combined left-sided and bilateral wounds, there were 7 deaths (36.8 per cent). Three of these involved the splenic flexure of the large bowel. The causes of death in the other 4 were: cerebral hemorrhage (embolism?); abscess in the region of a nephrectomy; and (bilateral case) massive hemorrhage from the liver in which both lobes were broken across in their long axis. Of the 36 right-sided wounds there were 2 deaths (5.5 per cent), one from hemoglobinuric nephrosis, and the other from bile peritonitis resulting from an undiscovered nick in the common duct at its junction with the pancreatic duct. There were 3 deaths (42.9 per cent) in the 7 patients with large bowel involvement. A similar experience was noted by Betts.[2] In his total of 29 thoracoabdominal wounds, there were 6 deaths (20.7 per cent). In his series there were 5 deaths in the left-sided wounds (27.7 per cent), and the large bowel was involved 8 times with 3 deaths (37.5 per cent). These mortalities are more than acceptable in view of Hurt's findings of 135 deaths (47.2 per cent) in the follow-up of 286 patients with large bowel injuries.[3]

The position of the foreign body in a penetrating thoracoabdominal wound must be accurately located. Anteroposterior and true lateral roentgenograms of the abdomen are essential. Careful examination of the wound will occasionally be quite revealing. For example, the odor of *Bacillus coli* in two left-sided wounds was preoperative evidence of large bowel involvement, and the probability of gross pleural contamination indicated early surgery. Omentum was seen in three wounds and pericardial fat in one. It is likely that the omentum and large bowel were sucked up through the torn diaphragm by respiratory action whereas the pericardial fat was apparently driven into the wound of exit. These findings may be helpful in establishing the direction of the missile.

Left-Sided Perforating Wounds.—Perforating wounds in the lower costal margin should always be examined and treated for the possibility of diaphragmatic involvement. If the wounding occurs during the expiratory phase, the anatomic position of the wounds in the chest wall may be quite cephalad and yet there may be a torn diaphragm and injured peritoneal organs. In one instance of laceration of the diaphragm and spleen, the wound of entrance was in the fourth interspace just medial to the left nipple and the wound of exit was in the seventh interspace in the left midaxillary line. Perforating wounds of this region should be prepared as expected thoracoabdominal wounds. It is a simple matter, when débriding these wounds, to enlarge the one nearest the diaphragm to permit examination of this muscle. The chest wall is relatively thin in this region and, consequently, the majority of wounds are sucking. In the process of closure, enlarging these wounds will permit an adequate check on the diaphragm. Involvement of the left diaphragm is of itself sufficient indication for thoracotomy. To reduce postoperative wound infection in diaphragmatic involvement, it is best, if anatomically feasible for good exposure, to resect a rib which is not in the wound of entrance or exit.

Left-Sided Penetrating Wounds.—Accurate localization of the missile, whether or not it is in the upper abdominal quadrant, is extremely important in left-sided penetrating wounds because its location determines the type of approach. Any damage done to intraperitoneal organs by a missile which enters the left upper quadrant and remains there can readily be reached and remedied by a transdiaphragmatic approach. This approach permits access to this region with greater technical ease than is attained through a laparotomy approach and the usual postoperative complications are decreased. However, if the missile passes into any other quadrant of the abdomen it is safer to perform a laparotomy.

The left transdiaphragmatic approach has many technical advantages which permit the following to be done with ease: splenectomy; exploration and repair of the entire stomach from the esophagogastric junction to the duodenum; examination and mobilization of the colon from the hepatic flexure to the beginning of the sigmoid loop; subcostal or midline exteriorization of the colon by either loop or spur colostomy; inspection and treatment by suture or resection and anastomosis of the small bowel from the ligament of Treitz to within a few inches of the cecum; and visualization and removal of the left kidney.

After anesthetization, the patient is placed on his right side with a folded blanket under the lower lateral portion of the right costal margin. A long strip of adhesive placed over the left hip and attached to the litter anteriorly and posteriorly fixes the patient in position. The left leg is flexed at the hip and knee with a pillow beneath the knee. With the right leg extended, the great saphenous vein anterior to the medial malleolus or at the medial border of the middle of the tibia is available for the administration of fluids during the operation. Removal of the ninth rib usually gives the best approach to the left upper quadrant. However, as this varies with habitus of the individual, the proper rib can be selected during débridement of the penetrating wound. The rib resection should stop short of the costal cartilage to prevent the occurrence of a chondritis if contamination is present. In opening the diaphragm, the wound of penetration should be included, if possible. When a splenectomy is indicated, the lateral peritoneal reflection over the spleen is easily cut, and the spleen is delivered into the opening in the diaphragm. The splenectomy is technically easier if, after delivery, the operator and the assistant change sides. The assistant is behind the patient and maintains the delivery of the spleen with his left hand. The operator, facing the patient, can then pass his fingers beneath the splenic pedicle, and this decreases the possibility of injury to the greater curvature of the stomach and the tail of the pancreas. In exploration of the stomach, opening of the gastrocolic omentum permits access to its posterior wall. For exposure of injuries in the region of the esophagogastric junction, especially for those on the lesser curvature side, it is necessary to cut the left triangular ligament and mobilize the left lobe of the liver. To expose the left kidney, the lateral peritoneal reflexion of the upper part of the descending colon and the lienocolic ligaments must be cut, and the colon laid mesially. This approach permits a nephrectomy to be done easily.

Two layers of interrupted silk are used to close the diaphragm. It is important that each suture of the first layer should include the peritoneum. There is no need to crush the phrenic nerve. The apparent defect in the lateral costal margin of the diaphragm is a result of contraction after severance of the attachments. It is repaired by suturing the diaphragm to an intercostal muscle one or more spaces higher than its original attachment. This principle of reattachment is important, for the original site is usually the area of the wound of the chest wall with its accompanying fractures and will not be stable enough for efficient diaphragmatic action. When the diaphragmatic crura are torn from the vertebral column, the first layer of sutures should be passed through the superior surface of the lobe of the liver. A sufficient number of the sutures will hold, fixing the liver against the undersurface of the diaphragm and preventing herniation.

Occasionally a left thoracoabdominal wound of the penetrating type occurs without hemo- and/or pneumothorax. The foreign body is in the left upper quadrant. The wound of entrance is not sucking because of its oblique course through the chest wall. The question arises: with the possibility of a large bowel injury, should the approach be through the pleural cavity in which there is no preoperative evidence of contamination? We have not settled this conclusively because the problem occurred in only two of our cases, and both were handled through a laparotomy with closure of the diaphragm from below. However, considering the technical ease of doing these operations transdiaphragmatically as compared to doing them through a laparotomy incision, we propose in the future to do them transdiaphragmatically. The factors of postoperative collapsed lung and pleural infection need not figure prominently in the decision if proper emphasis is placed on the toilet of the pleura and on pulmonary expansion during closure of the thoracotomy.

Right-Sided Perforating Wounds.—Right-sided perforating wounds involve the lower third of the chest and are usually at a right angle to the ribs. Careful alignment of the wounds of entrance and exit will assist in assuming which organs are involved. In our series perforating wounds of this region involved only the liver and diaphragm. These may be divided into two types according to position in relation to the diaphragm and to size: the "blowout" type (traumatic thoracotomy) through which the diaphragm and liver are readily exposed, and the small wound at a distance from the diaphragm and liver which requires the resection of a rib not involved in the wound to give the desired exposure. In the latter type, the rib to be resected can be selected through the débrided wound.

It is felt that right diaphragmatic lacerations should be closed and the infradiaphragmatic space should be drained subcostally. The diaphragmatic injuries are always associated with a liver torn to some extent. If the diaphragmatic rent is not repaired, a biliary fistula into the pleural cavity may occur. Biliary pleural fistulas are in themselves difficult to treat and there is probably more than a casual relationship between biliary-pleural fistulas and pleural empyemas. This is particularly true if the laceration of the diaphragm is over the bare area of the liver. This region does not have available peritoneum to include in the closure of the diaphragm. The pooling of undrained blood and necrotic material from a liver tear can more readily break through a sutured diaphragmatic rent unfortified by peritoneum. In the patients in whom the missile lacerates the diaphragm and the liver in the bare area region, drains can be laid over the rent in the liver, and, by breaking through the lateral reflection of the peritoneum over the right lobe of the liver, the drains can be exteriorized subcostally.

Right-Sided Penetrating Wounds.—Twenty-six of the 36 right-sided wounds in the series were of the penetrating type. Such cases pose the problem of determining whether the foreign body has passed entirely through the right lobe of the liver and is in the peritoneal cavity, or whether it has stopped in the liver. When it is evident by roentgenogram that the fragment has stopped in the liver, the management is as just detailed in right-sided perforating wounds. The problem is then merely one of drainage of the suprahepatic space and repair of the diaphragm. If the fragment is readily available in the liver, it is removed. Protracted exploration for a buried foreign body will often result in considerable hemorrhage.

When the missile is revealed to be in the peritoneal cavity, the abdomen is best explored through a laparotomy approach. Wounds of the diaphragm no higher than approximately the eighth interspace and no farther back than the posterior axillary line can be exposed and sutured from below. The condition of the patient will determine whether or not the diaphragm, penetrated in other areas, will be repaired. It is obvious that a fatality will not occur from an unrepaired right diaphragm. The likelihood of a biliary-pleural fistula occurring cannot be considered of major importance in the patient who is in poor condition because of intraperitoneal injury and in whom a laparotomy is necessary. In such instances, débridement, closure of the chest wound, insertion of an intercostal tube, and aspiration of all blood and air precede the laparotomy. These are done to establish and to maintain as high a vital capacity as possible during the laparotomy. Otherwise, blood from the liver draining into the pleural cavity may result in a considerably lowered vital capacity by the time the laparotomy is completed. The intercostal tube drains into a water-trap bottle and indicates the amount of liver bleeding. This procedure was necessary in only two of our patients, one of whom developed a biliary-pleural fistula. When the patient's physical status permits, a thoracotomy with repair of the diaphragm and expansion of the lung precedes the laparotomy and suprahepatic drainage. This amount of surgery was performed in the majority of cases in our series and was not prohibitive, but it is to be applied only after careful evaluation in each case.

INJURIES TO MEDIASTINAL STRUCTURES

Exploration for injury to mediastinal structures is done for one of two reasons: suspected injury to the esophagus, and continued bleeding from large vessels or from the heart. A small fragment whose position and course is such that neither of these is suspected need not be removed. Enlargement of the mediastinum by extravasation of blood as seen by roentgenogram does not merit exploration unless it is accompanied by signs of progressive swelling of the neck veins. We have learned that neck wounds also may present a markedly enlarged superior mediastinum. The enlargement can be caused by blood from a neck vessel dissecting into the mediastinum. Before proceeding with such extensive exposures as removal of a part of a clavicle and a portion of the sternum, it is well to investigate thoroughly the neck wound because the source of hemorrhage may be reached from above. The source of hemorrhage may be available by exposure in the base of the neck even when the missile is in the superior mediastinum. With the missile in this region not easily removed, the superior mediastinum can be drained through the cervical wound of exploration.

Diagnosis of injury to the thoracic esophagus is very difficult. Dysphagia was not noted in our series. However, severe back pain radiating down into the lumbar region was a constant complaint and was unrelieved by a large dose of morphine. There have been no constant pathognomonic roentgenographic findings. Air in the mediastinum was seen only once. The only way that mortality from esophageal injury could be reduced would be by doing a thoracotomy on every suspected injury. Accurate preoperative diagnosis is far from the rule. The picture is always obscured by the hemopneumothorax hiding the missile's unpredictable path in traversing the lung. However, keeping in mind the exact position of the esophagus in the various portions of the thorax in relation to the position of the missile as determined by accurate anteroposterior and lateral roentgenograms will be of some help. But even this help is not available when the missile passes through the mediastinum.

The basic principles of treating lacerations of the thoracic esophagus are repair of continuity, decompression by posterior paravertebral mediastinotomy, and diversion of subsequent infection from the pleural cavity by repair of the mediastinal pleura. The esophageal repair is often difficult, and some may leak even when well repaired. In others, the position of the laceration, such as at the level of the arch of the aorta, prevents adequate exposure and may be left unsutured. All these possibilities are covered by establishing a mediastinotomy and closing the rent in the mediastinal pleura. The approach should be a posterolateral transpleural one as close to the level of the suspected injury as possible rather than that of a posterior mediastinotomy, for the latter is too limited in its exposure for associated hilar and mediastinal injuries. The esophagus, especially the upper third, is best approached from the right side, but, when the wound of entrance and the missile are both on the left, no choice is afforded. Closure of the esophagus is done in its transverse axis and should consist of two layers. A continuous fine silk suture is used for the inner layer and interrupted silk mattress sutures for the outer ones. Care should be used to make certain that the two layers are distinct in that the ends of the inner layer of sutures do not protrude into or through the outer layer. If the Levine tube cannot be passed preoperatively, it may be introduced and guided past the injury during the repair. The mediastinotomy is performed during the thoracotomy in the following manner: the mediastinal pleura is dissected for a considerable distance in all directions; a three-inch needle is slid alongside the vertebral body in the mediastinum at the level of the injury—the needle point is pushed out of the skin posteriorly and the hub of the needle is left in the intercostal space at the level of the esophageal rent; a Penrose drain is placed alongside the laceration *and packed down to the intercostal space alongside the* body of the vertebra; the mediastinal pleura is then closed. When a pleural defect makes closure difficult, a flap is raised from the parietal pleura and the closure is accomplished without tension. The remainder of the posterior paravertebral mediastinotomy is done using the needle as a guide to remain extrapleural and to lead directly into the mediastinum to the drain. Segments of ribs, transverse processes, and intercostal muscle bundles are resected above and below the needle. The end of the drain is exteriorized. It is well to remember that mediastinotomy wounds close rapidly. Therefore, gauze should be inserted to keep the wound edges and the intercostal space open.

We found two types of hemorrhage from the cardiac chambers: active and "controlled." An example of active hemorrhage was found in a patient in whom no tamponade existed because of decompression by laceration of the pericardium. A picture was presented of massive hemothorax in which continuing hemorrhage was suspected. At operation, a widely lacerated pericardium was found, and a laceration of the myocardium was discovered as the source of hemorrhage. "Controlled" hemorrhage was typified by a case in which the missile entered the posterolateral aspect of the junction of the right auricle and the superior vena cava. The pressure of the resulting tamponade proceeded only to the point of cessation of the hemorrhage. Opening the pericardium relieved the tamponade, and the hemorrhage did not recur. Both patients made an uneventful recovery. "Controlled" hemorrhage raises the problem of the use of pericardial aspiration to replace operative treatment. It is of aid in preoperative management, but in tamponade caused by irregular fragments it would be hazardous to depend on this type of treatment alone in all such cases. Repeated aspiration may be sufficient in some patients but it would appear to be safer to operate on any patient in whom tamponade reappears after two aspirations.

We had two patients with hemorrhage from large mediastinal vessels. One had lacerations of the hemiazygos and accessory hemiazygos veins and the other had two small holes in the thoracic aorta. Surgical intervention in the latter was quite unsuccessful.

CHEST WALL WOUNDS

Chest wall wounds without penetration of the pleura seldom present major problems. In 64 chest wall wounds, we found 6 (9.4 per cent) instances of associated pneumothorax. These pneumothoraces responded to simple thoracentesis. There were 12 (18.8 per cent) instances of contusion of the lung underlying the chest wall wound. These hemorrhagic contusions all cleared without sequelae in nine to twelve days. Considering this incidence of pneumothorax and contusions of the lung, it is suggested that roentgenograms be made for all chest wall wounds.

Another problem that occasionally occurs is the involvement of the subscapular space. There were 6 (9.4 per cent) such lesions in our series. In wounds traversing the subscapular space, the possibility of anaerobic gas infection should be kept in mind. To do a complete débridement, an incision approaching the magnitude necessary for a thoracoplasty and mobilization of the scapula would be necessary. This was not done in these cases. Instead, incisions were made along the track of the missile at the vertebral and axillary borders of the scapula, and injured muscle and foreign material were removed. Through-and-through Penrose drains were inserted. A dependent drain was placed in the subscapular space through an incision in the triangle of auscultation at the inferior angle of the scapula. No gas infection developed in these patients. All such patients should be kept at least 6 days postoperatively. At any rise in pulse rate, occurrence of pain, or change in the patient's demeanor, the wound should be examined immediately. Missiles remaining in the subscapular space should be removed and thorough drainage instituted. These precautions should eliminate or at least reduce the incidences of subscapular abscesses. Elimination of the possibility of these abscesses is important, for they may go undetected for long periods.

COMBINED THORACIC WOUNDS AND SPINAL CORD INJURIES

In this series, four patients with chest wounds had associated spinal cord injuries. One had a penetrating wound of the chest in which the missile was pressing on the cord. Three had perforating wounds with a traumatized spinal cord. In débridement and exposure to close the adjacent sucking wounds, removal of fragments of the laminae disclosed the extent of the cord injury. In two, the cord was markedly swollen but intact; in one, the cord was the site of extensive hemorrhage; and in the last, the cord was transected. All cases presented the picture of transverse myelitis.

The mortality was in direct relation to the skin anesthesia level. The two patients who died had skin anesthesia above the umbilicus (one just below the nipples and the other at the xiphoid). The two who lived had skin anesthesia levels below the umbilicus (one just below the umbilicus and one halfway between the umbilicus and symphysis pubis). All four patients had approximately the same amount of lung and pleural damage. The two deaths occurred four or five days following operation from pulmonary edema that did not respond to treatment. Positive pressure oxygen therapy and endotracheal suction were of no avail. Apparently, paralysis of the abdominal rectus muscles reduces the efficiency of the cough mechanism and may be a factor in permitting pulmonary edema to ensue in the acutely injured. The tone of the recti in a gentle cough or actual recti contraction during a vigorous cough gives the diaphragm an opposing intra-abdominal pressure. When the opposing pressure is lost by paralysis of the recti, the cough becomes ineffective because the expulsive action merely bulges out the abdomen. Possibly a snug abdominal binder or a plaster abdominal jacket for the first four or five days may be efficacious in replacing the function of the recti. With the innervation of only the upper half of the recti intact, the cough mechanism can function efficiently. From these few cases, it seems that a patient with a penetrating or perforating injury to the lung and injury to the spinal cord has a poor prognosis if the skin anesthesia level is above the umbilicus.

PRINCIPLES OF PULMONARY EXPANSION

Originally, drainage was carried out intercostally in any patient with lacerations of the lung, contamination of the pleural cavity, or abrasions of the parietal pleura. The intercostal tubes usually functioned for only two days. Aspiration was then necessary. Recently, following the suggestion of Betts and Lees,[2, 4] thorough pleural lavage was done in addition to expanding the lung completely at the time of operation, thus decreasing the use of intercostal drainage. Lung expansion has been quite satisfactory and thoracentesis has been done less frequently. The recent change from drainage to nondrainage is not as important as the factors of *thorough pleural lavage and complete expansion of the lung at the time of closure.* If these two procedures are carried out conscientiously obliteration of the pleural space has a good start. Any pleural fluid which then forms is of a serous type that lends itself to aspiration more easily than does a grossly bloody fluid containing clots. Adherence to these principles may eliminate total empyema and should reduce the percentage of organizing fibrothorax. We do not recommend that the generally accepted principle of intercostal drainage in military surgery be revised but in light of our experience during the past few months we feel its application in such cases need not be practiced routinely. *Whether or not a drain is used is not as important* as proper toilet of the pleura and complete expansion of the lung at the time of operation.

Frequently, during expansion of the lung by the anesthetist, we have noted that the lateral portion of the lower lobe did not expand as readily as the remainder of the lung. With the patient on his side, the partial collapse of the lung permits a folding over of this portion onto the diaphragmatic surface of the lower lobe. When it is pulled out and unfolded, aeration occurs.

POSTOPERATIVE CARE

Postoperative care should focus on the main objective of complete and early pulmonary expansion. In this respect, the operative and postoperative period should be considered as one. The goal, a completely expanded lung, is achieved at the time of closure of the wound, and is maintained by repeated thoracentesis when even small amounts of pleural air and/or fluid are present.

Maintenance of a clear tracheobronchial tree by an energetic cough routine is aided by intercostal nerve block when pain is an inhibitory factor, and is another step in maintaining aeration of the lung. When the airway is not cleared by these methods we resort to tracheal aspiration. Postoperative bronchoscopy is necessary and should not be delayed if these measures have failed. Sedation is kept at a minimum to insure prompt cooperation with the cough routine. Nasal oxygen is given for postoperative shock and is continued only in those patients in whom pulmonary moisture persists. When the need for oxygen continues, one may be certain that all of the available parenchyma is not being

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From the Preface

PNEUMOPERITONEUM as a therapeutic procedure has been known to the medical profession for over fifty years. In the early days of its employment it was used in the treatment of tuberculous peritonitis only. From this modest beginning its clinical application has been gradually expanding, as explained in the chapter, Historical Review.

During the past decade therapeutic pneumoperitoneum has been gaining a gradually increasing recognition and acceptance. Encouraging reports have been published in the medical literature on its value in the treatment of emphysema, bronchial asthma, pulmonary abscess, pulmonary hemorrhage, and particularly on its employment in the treatment of tuberculous peritonitis and enterocolitis, and of pulmonary tuberculosis. Furthermore, satisfactory observations have been reported with this treatment in the management of a number of other conditions which are discussed in detail in the chapter on the miscellaneous applications of this method.

Pneumoperitoneum is a technically simple and safe procedure. Every physician with average manual dexterity should be able to give this treatment. As a matter of fact, it has been proposed, and I believe justly so, that pneumoperitoneum treatment of ambulatory patients should be carried out by general practitioners whenever given circumstances so require. *I maintain that reading over carefully the outlines of its technique should enable one to give pneumoperitoneum without any difficulty.*

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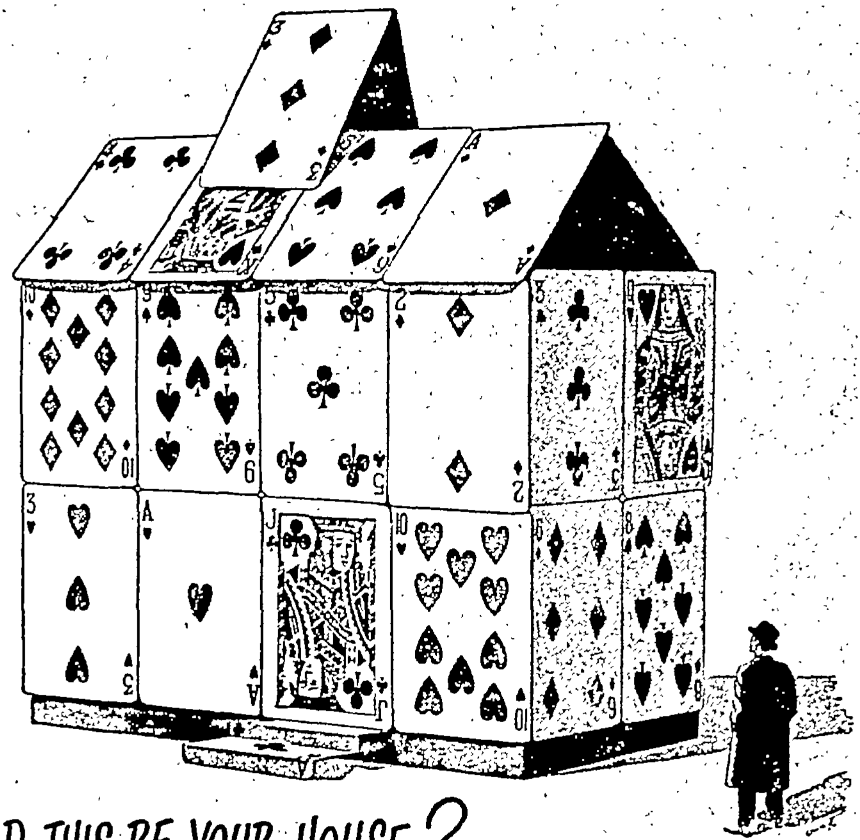

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Vol. 13 JUNE, 1944 No. 3

The Journal of Thoracic Surgery

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The Journal of Thoracic Surgery

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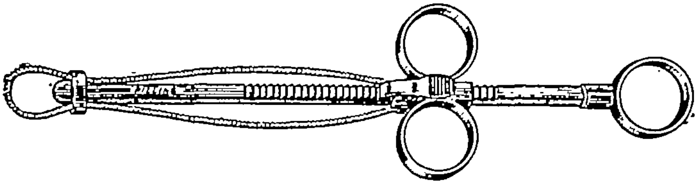

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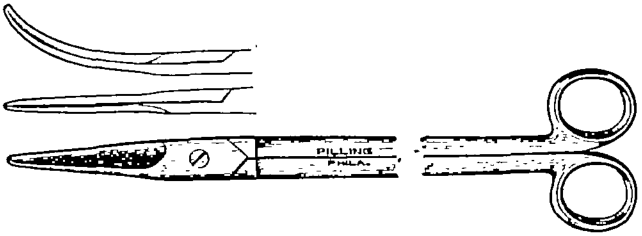

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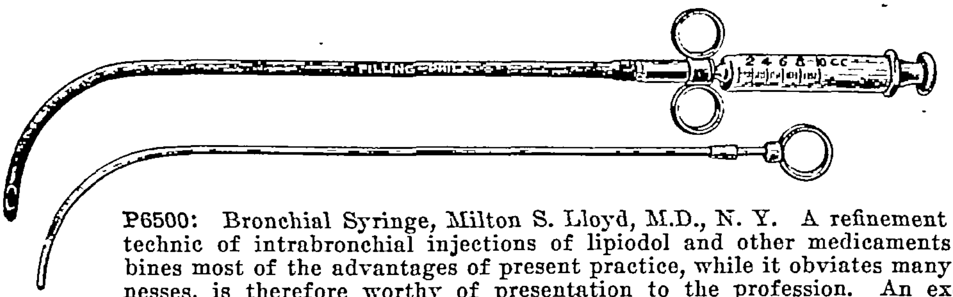

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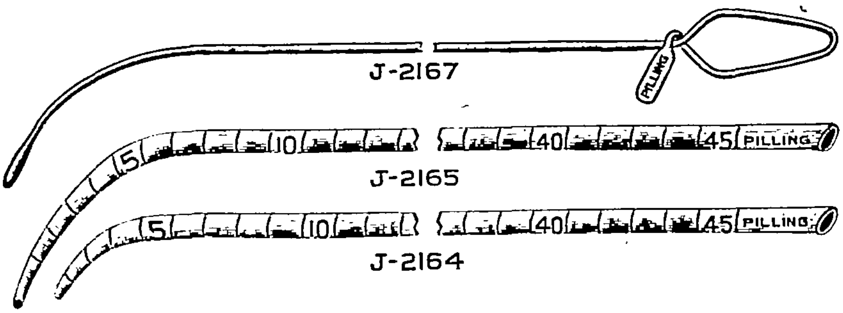

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The Journal of
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Vol. 13 June, 1944 No. 3

Original Communications

DERMOID CYSTS AND TERATOMATA OF THE MEDIASTINUM

A Review

N. Lloyd Rusby, M.A., D.M. (Oxon), M.R.C.P. (London)
London, Eng.

"It would be difficult to find in the adult human body a region of less intrinsic interest than that space between spaces known to anatomists as the anterior mediastinum." Thus spoke J. C. Wilson of Philadelphia at the beginning of a lecture on the differential diagnosis of mediastinal tumours in 1884. However unpretentious this "space between spaces" may appear to the anatomist, it does not lack appeal to the clinician and pathologist for one group of tumours found therein, the dermoid cyst and its more complex blood relation the teratoma, has provided for more than a century a fascinating field for speculation and study. Such tumours are found uncommonly in this situation; an estimate of their rarity can be formed from the publication of Hare (1889), who collected from the literature 500 cases of mediastinal disease of which only 10 were tumours of this type. Moir (1936) has stated that only two examples were among the patients admitted to the general medical wards of the Leeds Infirmary over a period of ten years, while at the London Chest Hospital, which draws its material from all over the country, five cases have been seen in seven years. Although rare, they are not unimportant both with regard to the manifestations to which they give rise and to the treatment that they require.

HISTORICAL SURVEY

The first example of a tumour of this type to be recorded, a dermoid cyst of the mediastinum, was made the subject of an address to the Medico-Chirurgical Society of London on Nov. 25, 1823, by J. A. Gordon,

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From the London Chest Hospital, Victoria Park, England.

Received for publication Sept. 23, 1943.

and subsequently published by him in the Transactions of the Society in 1827. It concerned a young woman, aged 21 years, seen at the Islington Dispensary, where the diagnosis of pneumonia was made. The illness pursued an atypical course for, despite repeated blood letting, she was left with an irritating cough and a persistent tachycardia of 120 beats per minute. The appearance, later, of a small pulsating tumour beneath the sternal end of the left clavicle caused a revision of the diagnosis in favour of an aneurysm of the aorta or innominate artery. This swelling increased in size and burst to discharge a serous fluid. The girl died shortly afterward and a limited autopsy disclosed an adherent tumour of the anterior mediastinum containing sebaceous matter and hair. At one end of the cyst was a plaque of bone that bore a striking resemblance to the superior maxilla and to which seven teeth were attached.

Spath (1836) reported a large tumour of the anterior mediastinum extending into the neck. It was discovered post mortem and was composed of fibrous and fatty tissues, bone, and cartilage, but no microscopic study of the specimen was undertaken. Three years later, Munz (1839) published a detailed clinical account of a dermoid cyst' in a woman of 28 years who had suffered all her life from catarrhal affections and who had noticed hairs in her phlegm since she was 15 years old. She became phthisical and died, the coexistence of pulmonary tuberculosis and a dermoid cyst being confirmed at autopsy. This case is quoted by Godlee (1889).

In succession to Büchner's case in 1853, sporadic examples were reported by later writers (Lebert, 1858; Cordes, 1859; Cloetta, 1861; Salomonsen, 1864; Collenberg, 1869) until 1869 when the condition was clearly recognised as a clinical entity, for in that year Nobiling showed a preparation of a cyst that had been correctly diagnosed during life, owing to the expectoration of hairs. In the remaining years of the nineteenth century, thirty-five additional cases were recorded, serving mainly to enrich knowledge of the pathology of the condition and to emphasise the difficulty in reaching an accurate diagnosis of the condition during life and before operation. Reviews of the subject during this period were presented by Hare (1889), Dardignac (1894), Pflanz (1896), and Ekehorn (1898).

Dangschat, in 1903, collected forty-two examples from the literature and added two of his own, while two years later Morris (1905) placed the total number of cases on record at fifty-nine; these included a patient that he had himself observed and who had been operated upon by Senn (1905). Morris, however, omitted one or two references that have come to light from other sources and included one case (Fletcher, 1897) of a congenital cyst arising in connexion with the foetal oesophagus that should be classed separately.

Hertzler (1916) published an exhaustive review of the literature up to that time which comprised seventy-two case reports, and Lambert and Knox, in 1920, reported six cases. Of these only one was a proved teratoma but four others are sufficiently suggestive on clinical grounds to be accepted as genuine examples of this condition, while one case with a syphilitic history, a doubtfully positive Wassermann reaction, and a posteriorly placed tumour cannot justifiably be included.

Kerr and Warfield, in 1928, computed the total number of recorded cases at 138, but a study of the original case reports has raised a doubt as to whether some of these are, in reality, genuine examples of intrathoracic dermoid cysts or teratomata. For instance, the cases published by Clutton (1887) and Selby (1906) would seem to resemble presternal dermoids having no connexion at all with the mediastinum. Again, a reading of Fawcett's (1906) original account of a woman, aged 62 years, whose symptoms had been of short duration and were correlated with physical signs pointing to bronchial obstruction strongly suggests, in the light of the post-mortem findings and in the absence of microscopic proof to the contrary, a diagnosis of a breaking-down carcinoma of the right lower lobe bronchus with secondary deposits in the mediastinal glands. Other examples could be quoted. Since Kerr and Warfield's article appeared, ninety well-authenticated cases of this condition were published up to the end of 1939. It is unprofitable at this time to pursue the detailed study of the literature much beyond the outbreak of the present war in view of the difficulty of obtaining access to continental literature. As far as possible the original source has been consulted for information or an abstract of it obtained, but this has not always been possible. After allowance has been made for those examples, included by other workers, which cannot be accepted on the evidence presented and excluding the six cases described for the first time in this communication, it has been possible to trace in the literature up to the end of 1939, 245 recorded cases of intrathoracic dermoid cysts and teratomata. It is upon practically the whole of this material that the following review is founded.

CASE REPORTS

CASE 1.—E. S. was a married woman, aged 42 years. Her past medical history had been uneventful and free from major chest affections, but she had noticed that for some years she had been unduly short of breath on effort and subject to attacks of palpitation. No more tangible symptoms than these became manifest, however, until towards the close of 1937, when she began to experience an intermittent pain in the centre of the chest about the level of the second rib which spread across to the left side. It was sharp in character and although spasmodic did not resemble a true anginal pain on effort. One week later she became aware of a small pulsating tumour in the neighbourhood of the second left rib a short distance from the sternum, and it was on account of this that she attended her local hospital. Physical examination suggested an aneurysm of the thoracic aorta and an x-ray picture showed a well-defined "tumour" in the centre of the chest that abutted slightly into both the right and left lung fields. The Wassermann reaction was negative.

She was confined to bed for three months and the symptoms improved; for a further eight months good progress was maintained. At the end of that time, however, she complained of pain across the shoulders and up and down the neck and noticed that her "heart was thumping a lot." She also developed a dry, croupy cough that was worse at night. She was seen by Dr. T. W. Preston who was not satisfied with the previous diagnosis and referred her to the London Chest Hospital for further investigation. At the time of her admission she suffered from a dragging pain in the left side of her chest, a short, dry, wheezy cough, often noisy and provoked by lying back, noticeable pulsation in the chest, shortness of breath on walking, and some recent loss of energy. There had been no voice change.

Physical Examination.—The most striking feature was the pulsation, which was of sufficient force to shake the whole body. Complexion was unhealthy but general bodily condition was good. There was an irritating dry cough that at times possessed the "gander" quality traditionally associated with aneurysm of the aorta. She always sat up in bed, knowing that to lie back would be to invite a fit of coughing.

The heart's impulse was forceful and the apex beat located in the sixth interspace one-half inch outside the midclavicular line. There was a localised area of pulsation in the second left interspace two and one-half inches from the midsternal line; over this area the percussion note was impaired and a systolic bruit heard. There was unmistakable tracheal tugging. Blood pressure was 135/85 mm. Hg and approximately equal in both arms.

The left side of the chest was slightly fuller in its upper part than the right and in movement lagged behind its fellow. Air entry into the left apex was poor. There were no other abnormal physical signs in the chest.

The pupils were regular with a moderate aperture and reacted briskly to light and convergence. All tendon jerks were normal. No other significant feature was found.

Radiography.—An anteroposterior picture of the chest (Fig. 1) showed that the tumour had increased in size since the previous film was taken a year before. The shape and clear-cut edge, however, remained the same. A left lateral picture (Fig. 2) disclosed the position of the tumour in the anterior mediastinum beneath the sternum. Fluoroscopic screening demonstrated the vigorous pulsation of the mass. The aorta was investigated in more detail; the transverse arch and descending thoracic aorta could be visualised with ease and there was no widening or any other detectable abnormality in this portion of the vessel. It was not possible, however, to make out the first part of the ascending aorta, and no opinion could be formed as to whether it was healthy or diseased. A series of tomographic pictures was unhelpful.

Other Investigations.—Wassermann reaction was negative before and after a provocative dose of novarsenobillon. Electrocardiograph showed a normal rhythm with a right axis deviation. Friedman test, Casoni reaction, and complement fixation test for hydatid disease were all negative. There was no abnormality in the urine. Blood count: red cells 4,700,000 per c.mm. with 92 per cent haemoglobin, leucocytes 15,000 per c.mm.; blood sedimentation rate 24 mm. in the first hour (normal from 3 to 7). The slightly raised leucocyte count and the elevated sedimentation rate suggested the presence of infection. There was no fever but a sustained tachycardia of 100 to 120 beats per minute and a respiration rate of 22 to 25 per minute were abnormal features.

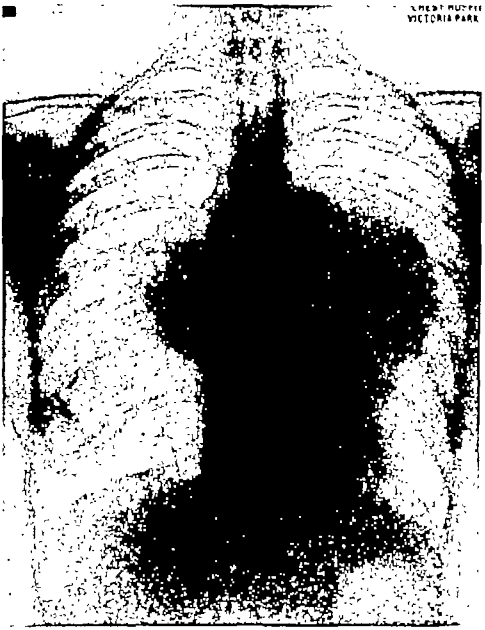

Fig. 1.—Case 1 (E. S.). Anteroposterior skiagram of the chest.

An accurate clinical diagnosis was not easy; a saccular aneurysm of the ascending aorta or a benign tumour of the mediastinum, probably a dermoid cyst, seemed the most likely possibilities. The real difficulty sometimes encountered in making a distinction between these two, at first sight apparently clear cut, conditions will be discussed more freely later. It seemed that in this instance a benign tumour of the mediastinum transmitting pulsation was the more likely of the two alternatives and, as the tumour was embarrassing the patient and threatened her life in the near future if its steady growth was maintained, an exploratory thoracotomy was advised and accepted.

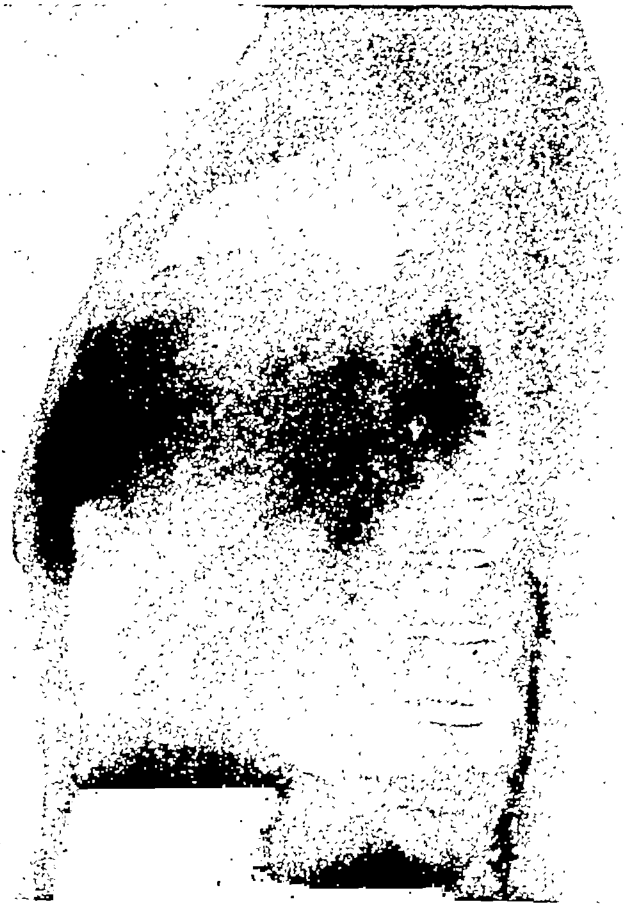

Fig. 2.—Case 1 (E. S.) A left lateral x-ray picture; the tumour occupied the anterior compartment of the mediastinum beneath the sternum.

Operation.—An artificial pneumothorax was induced on the left side as a preliminary to operation. The lung collapsed well, the localised area of pulsation became less apparent, and the percussion note over it resonant. The operation was undertaken under light gas and oxygen anaesthesia, but the patient died suddenly after the skin incision had been made and before the chest had been opened.

Autopsy.—A very large tumour was exposed as soon as the sternum was removed. It completely covered the heart and was adherent to the right and the left lung and, higher up still, to the trachea. It was impossible to separate the tumour from neighbouring structures and the thoracic viscera were removed with it.

The tumour comprised one large cavity filled with thick, yellow, greasy, sebaceous material containing many hairs. To the apex of the heart was attached a small daughter cyst, unconnected with the parent cavity but containing similar sebaceous material lacking hairs. The specimen is shown in Fig. 3. It was opened and as much of the contents as possible removed, but flakes of the yellow material clung to the posterior wall and to the hairs. The daughter cyst is shown below on the heart's apex and tags of lung tissue, firmly bound to the cyst, can be seen on either side. The wall of the cyst was moderately thick and uniform in contour except in the anterior portion along which a ridge ran from left to right and downwards across the surface; it was from this ridge, and only from this ridge, that the hairs sprang.

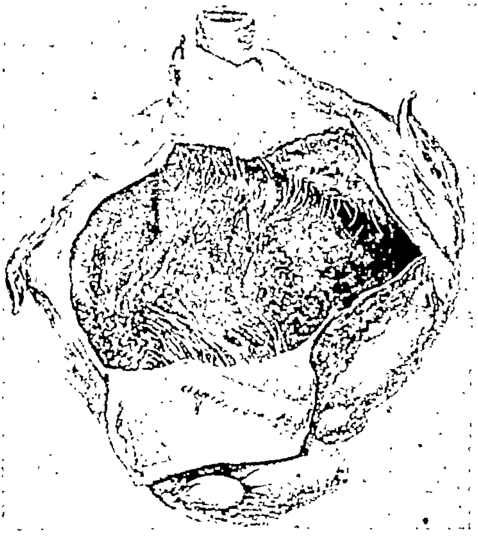

Fig. 3.—Case 1 (E. S.). Specimen removed at autopsy showing the dermoid cyst (opened) lying in front of the heart. The trachea is seen above, tags of lung at either side with the heart's apex, with small daughter cyst attached, below. (A photograph of a coloured drawing made by W. Thornton Shiells.)

A remarkable feature of the cyst was its firm union to the pericardium. A careful dissection of the tumour and surrounding structures was made, but it was impossible to find a line of natural cleavage between the posterior wall and the pericardium. This is very likely significant in explaining the persistent tachycardia, the unusual degree of pulsation, and the right axis deviation in the electrocardiograph, as the right ventricle was the chamber that bore the brunt of the additional burden, and contributed, perhaps, to the sudden and unexpected death of the patient.

Microscopy.—The wall of the cyst was composed of broad strands of fibrous tissue and some area of smooth muscle could be made out. The *inner lining consisted of stratified squamous epithelium with flaky* epidermis attached to it. Immediately beneath the surface, sebaceous glands could be seen in profusion together with their ducts, while deeper in the section were found many fat spaces and one or two plaques of cartilage.

The structure of this cyst proved it to be a teratoma of the dermoid type.

CASE 2.—D. W., aged 34 years, was a married woman. She had had *no major illnesses in the past and no liability to catarrhal affections.* She married a soldier and accompanied him to Egypt in 1931, where she stayed for two years. During this period she had an attack of dysentery of moderate severity for which treatment by injections was not advocated. Her husband's regiment was transferred to India, and she went with him and remained there for four years.

It was towards the close of her visit to India, in June, 1937, that the patient experienced the first symptoms referable to her present malady. These were pain in her chest and shortness of breath. The pain was felt in front of the chest and towards the left side and was provoked by taking a deep breath, by coughing, and laughing. It was sharp in character and, although intermittent, the patient was conscious of there being "always something there." At the same time she became aware of shortness of breath on walking, which impressed her as being unusual even when allowance was made for the fact that she was resident at a hill station. Five months later, the pain became worse and for ten days she felt it under the left armpit and remarked that it travelled down the inner side of the left arm to the fingers. Although there was no loss of power in the left arm she wanted to rest it all the time. Her general health was not good and she lacked energy.

She returned home from India in December, 1937, and on the journey had three haemoptyses consisting of bright blood mixed with a little sputum. Apart from this, there was little change in her symptoms until February, 1938, when a dry "tickling" cough became noticeable and further haemoptyses occurred. An x-ray picture was taken of her chest, blood tests were carried out, and the sputum was repeatedly examined for tubercle bacilli but none was found. Lassitude became more striking and the cough persistent, accompanied by a little mucoid sputum, until April, 1938, when she was admitted to a Military Hospital with an attack of "acute bronchitis." She had a temperature up to 102° F., cough and sputum increased, and the phlegm became thicker and resembled "anchovy sauce," according to her doctor. The sputum was not greasy and contained neither particles nor hairs. The illness lasted fourteen days and then abated but residual symptoms remained. The patient was transferred to the London Chest Hospital under the care of Dr. H. V. Morlock in December, 1938, for further investigation with a provisional diagnosis of an aneurysm of the aorta or pulmonary artery.

On admission to the hospital she suffered from a persistent cough productive of scanty mucoid sputum. The phlegm was never purulent and no particles or hairs were seen in it. There had been no haemoptysis for eight months. The pain in the chest of which she had complained for the past eighteen months was still present and associated with palpitations, especially when she became excited. Her breath was short when she climbed stairs and during bouts of palpitation.

Physical Examination.—The patient was a well-nourished woman with a normal temperature, pulse, and respiration rate. She lay comfortably in bed.

Movement of the left side of her chest was defective and the percussion note impaired over the anterior and upper part and a few crepitations were audible near the left edge of the sternum. There was no clubbing of the fingers.

A visible and palpable area of pulsation could be observed over the second and third left interspaces, but in all other respects the cardiovascular system was normal, the heart sounds were clear and precise, and the position of the apex beat was not displaced. The blood pressure was 115/80 and equal in the two arms. No tracheal tugging could be detected and the pupils were equal and regular, reacting normally to light and convergence. No enlarged glands were palpable and the other systems of the body presented no unusual features.

Radiology.—An anteroposterior and a left lateral x-ray picture showed a moderate-sized tumour abutting towards the left side and lying in the anterior mediastinum. The tumour possessed a clear-cut outline and the aortic knuckle could be easily identified (Figs. 4 and 5). A screening examination combined with a kymograph showed that the mass pulsated but was unconnected with the aorta and it was therefore concluded that this pulsation was transmitted rather then inherent.

Other Investigations.—The Wassermann reaction and the Kahn test of the blood were both negative.

The red blood cells numbered 5,500,000 per c.mm. with 94 per cent of haemoglobin; the white cells were 11,500 per c.mm. with a normal differential and no eosinophils. The blood sedimentation rate was normal.

The sputum contained a mixed flora, but tubercle bacilli and spirochaetes were absent from it.

Bronchoscopy supplied no helpful information.

An artificial pneumothorax was induced on the left side and the tumour inspected with a thoracoscope. It was rounded with a smooth wall and lay above and in front of the heart, pulsating freely with it. It had a cystic appearance.

The possibility of mediastinal hydatid cyst was considered and much interest was aroused when the Casoni test proved to be positive, but the hydatid complement fixation test, which was carried out later, was negative.

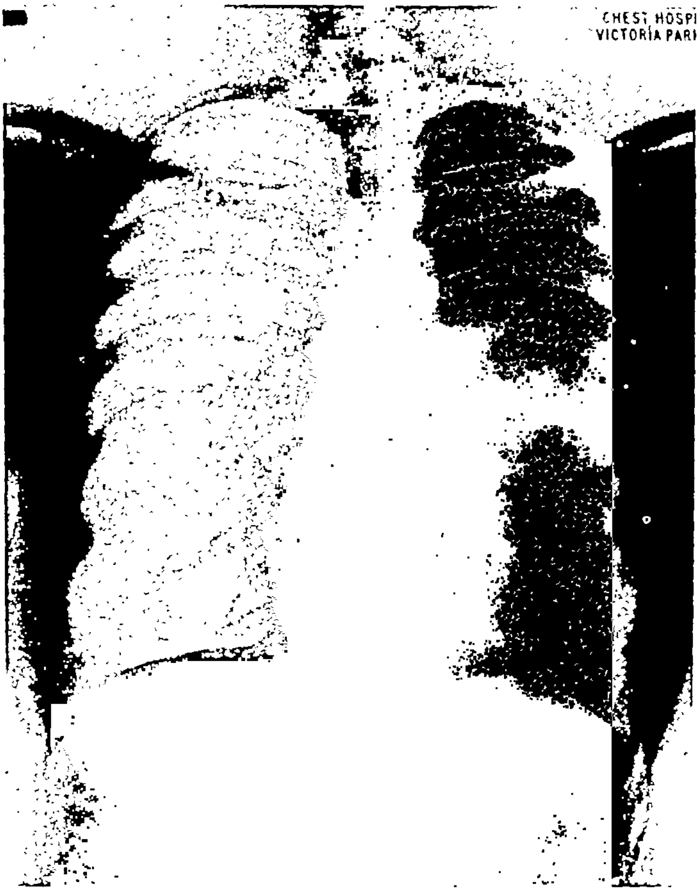

Fig. 4.—Case 2 (D. W.). Anteroposterior skiagram showing the tumour abutting toward the left side.

The evidence, therefore, pointed to a benign tumour of the mediastinum probably of the dermoid type, and a thoracotomy was advised and accepted by the patient.

Operation.—Operation was undertaken after the pneumothorax had re-expanded, and was performed by Mr. T. Holmes Sellors with an anterior approach under gas and oxygen anaesthesia.

The chest was entered through an intercostal incision; the divided sternal ends of the second and third ribs were retracted upwards and the fourth downwards. The tumour then came into view. The pleural capsule was incised and the tumour separated from the left lung and pericardium to both of which it was firmly united. Some difficulty was experienced in freeing it from the posteromedial attachment, especially at its upper pole which lay to the right of the midline, and an indefinite pedicle was seen. In the course of this manipulation the cyst wall was punctured and a white semisolid structure, the size of a thumbnail, herniated through the opening. To this some hairs were attached. The tumour was removed completely and the chest repaired without drainage.

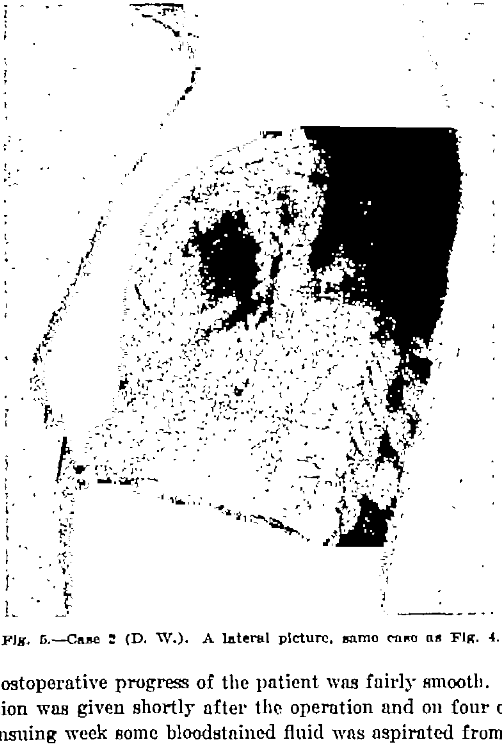

Fig. 5.—Case 2 (D. W.). A lateral picture, same case as Fig. 4.

The postoperative progress of the patient was fairly smooth. A blood transfusion was given shortly after the operation and on four occasions in the ensuing week some bloodstained fluid was aspirated from the left chest. No empyema developed and a thin trickle of serous discharge continued for some weeks from the sternal end of the wound, but this dried up and she was discharged perfectly well two months after operation.

The tumour was a multilocular cyst with thin walls containing a viscid, transparent, gelatinous fluid. There was a central core of warty consistence with one long hair and numerous short ones growing from it.

Microscopy.—The warty central core was made up of fibrous and fatty tissue and covered by squamous epithelium. Sebaceous glands could be seen deeper in the section and some of the ducts into which they drained could be picked out. The walls of the loculations were of similar structure. In one area some tissue closely resembling salivary gland tissue was identified.

This tumour, like the preceding example, could be classified as a teratoma of the dermoid type and exhibited, histologically, similar characteristics to many such cysts recorded in the literature.

CASE 3.—Mrs. Y., aged 25 years, was a patient under the care of the late Dr. A. J. Scott Pinchin. Her health, until 1934, had been unremarkable except for a tendency to catch cold, which "colds" always descended to her chest. In 1934, she had a more severe respiratory infection than usual during the course of which a heart murmur was noticed and she subsequently experienced breathlessness on going up stairs or when hurrying. A skiagram, taken early in 1935, revealed an abnormal shadow. Towards the end of that year the patient had a series of attacks of pain in the upper part of the left side of the chest associated with a temperature up to 100° F., much shortness of breath, and vomiting. She experienced three such attacks, each lasting about one hour. There were bouts of fever in between these attacks but little coughing, and her weight was steady.

She was well nourished and displayed few physical signs on examination of her chest. A radiologic investigation, however, disclosed a rounded mass with a well-defined edge lying in the anterior compartment of the mediastinum. The appearance of the tumour in the anteroposterior view is shown in Fig. 6; the lateral is not reproduced.

She was kept under observation for six months and during this period her symptoms increased in severity and the tumour in size, and she developed an effusion at the left base. The diagnosis of probable dermoid cyst was made and exploratory thoracotomy advised and accepted.

The operation was performed by Mr. T. Holmes Sellors, through an anterior incision, and adequate exposure of the mediastinum was obtained by resection of short lengths of the second and third ribs on the left side. An oval cyst, measuring about three and one-half inches in diameter, was seen lying in the anterior mediastinum. It was adherent to mediastinum and chest wall and, although it proved possible partially to free the cyst, its complete removal could not be effected in view of the firmness of the adhesion to the pericardium and aortic arch. Five-sixths of the tumour, however, was excised and sufficient mobilisation of the remainder was accomplished to enable it to be drawn up to the original incision and marsupialised to it. The fluid content of the cyst, which escaped under pressure, was the oily, sebaceous matter characteristic of the condition and contained hairs and cholesterin crystals.

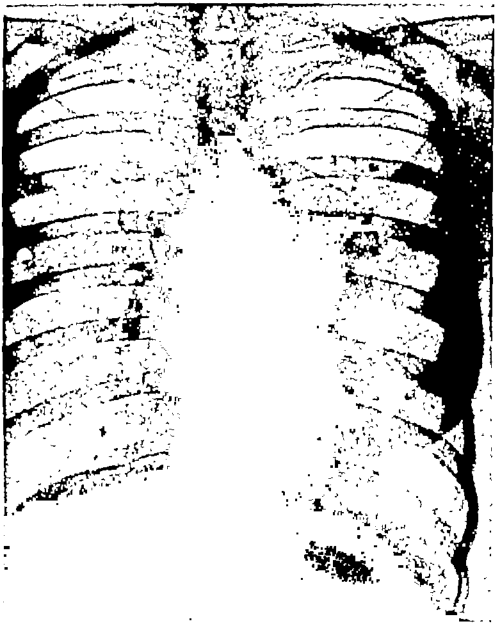

Fig. 6.—Case 3 (Mrs. Y.). Anteroposterior skiagram to show the size and shape of the tumour.

The patient's recovery from the operation was uneventful and all symptoms were relieved. There remained, however, a persistent sinus and a small amount of discharge. Despite this disability she gave birth to a male child whom she fed at the breast. Some months later, the sinus was treated with pure carbolic and formalin following which it closed and has remained sealed. This patient, when seen and examined six years after operation, was well with no symptoms, the sinus remained firm, and the x-ray picture showed little departure from the normal except for elevation of the left dome of the diaphragm due to interruption of the phrenic nerve at the time of operation.

This case, which again exemplifies the tendency of the dermoid cyst to enlarge and so cause symptoms early in adult life, is an excellent illustration of the success that may result from partial removal and marsupialisation if total extirpation is prevented by firm union of the tumour with neighbouring structures.

Case 4.—W. B. was a man, aged 33 years. He gave no history of any pulmonary disorder nor was he complaining, at the time he was examined, of any symptoms referable to the respiratory tract. One brother had recently contracted pulmonary tuberculosis and another had died of the disease a few years before, and it was in response to a request from Dr. J. E. Stokes, the Tuberculosis Officer of his district, that he attended the Dispensary as a "contact." A routine screening of his chest was performed and an unusual shadow seen. He was referred to Dr. W. Burton Wood's outpatient clinic at the London Chest Hospital for a more detailed investigation. This was in 1937.

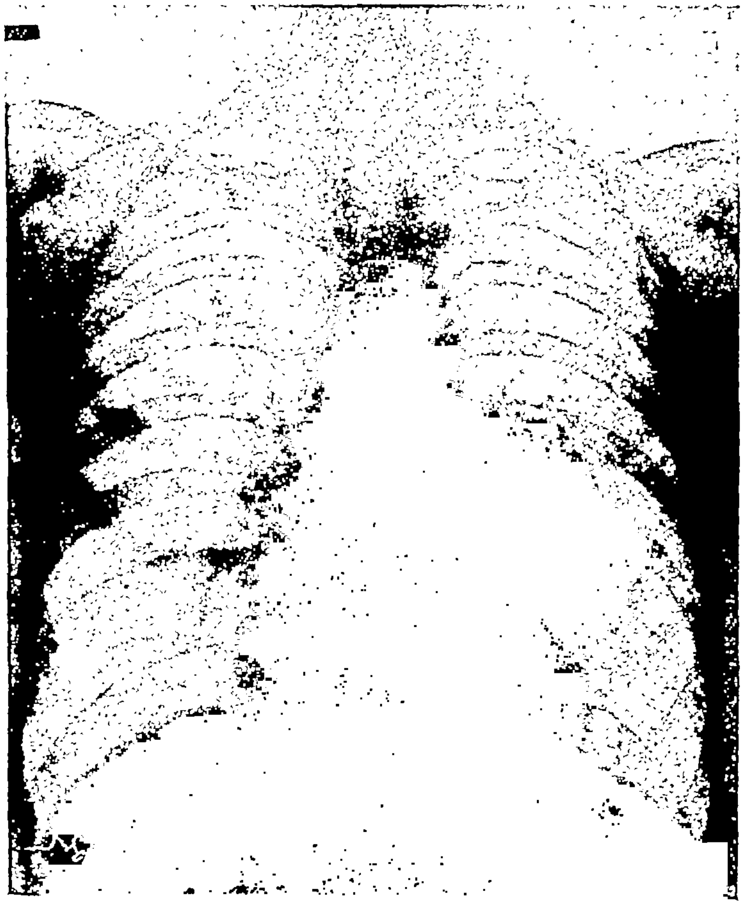

Fig. 7.—Case 4 (L. B.). Anteroposterior skiagram of a tumour of the left side, presumed to be a dermoid cyst.

He was a thin man of average build and his general physical condition was good. He confessed to no pulmonary symptoms and the only abnormal physical signs that could be detected were some impairment of the percussion note in front in the upper half of the left side of the chest and an area of localised pulsation in the third left intercostal space, one and one-half to two inches from the edge of the sternum.

The radiographic appearance of the chest in anteroposterior and left lateral view is depicted in Figs. 7 and 8. These films show a rounded shadow with a clearly defined edge apparently arising from the mediastinum and encroaching upon the left lung. It was anterior in position. On the screen it was separate from both the aorta and pulmonary artery as far as could be determined, and it pulsated.

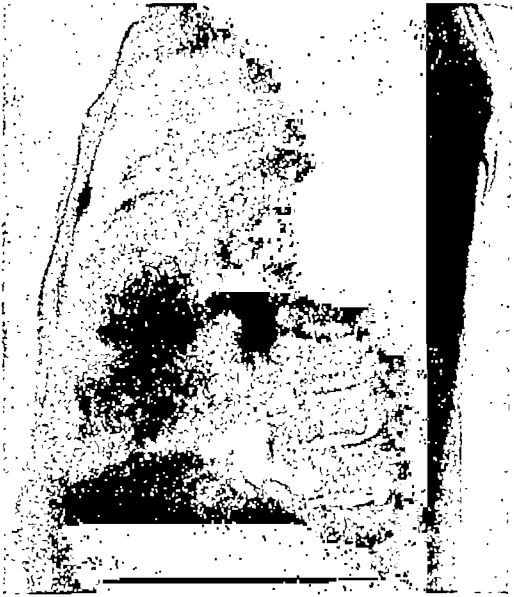

FIG. 8.—Case 4 (L. B.). Lateral view, same case as Fig. 7. The tumour is seen to lie in the anterior mediastinum.

The Wassermann reaction of the blood was twice negative, as was the Friedman test.

The patient was re-examined some months later when further x-ray pictures were taken. There had been no increase in the size of the tumour in that period.

He was encouraged to attend the outpatient department at regular intervals in order that a watch might be kept upon his progress, but his visits were only sporadic and later ceased entirely. All further attempts to get in touch with him were unsuccessful until December, 1939, when he was traced and persuaded to submit to another x-ray picture. The appearance of the tumour then, two years after the discovery of the disease, was similar in every respect to that previously noted and no increase in its size was detectable. At this examination, however, the impression was formed that the pulsation in the third left interspace was more obvious than it had been a year before.

The patient's mentality was of a low order and his reaction to a discussion as to the nature of his illness and what could be achieved and possibly prevented by surgery was unhelpful. For this reason and because of the symptomless character and stationary size of the tumour, an exploratory thoracotomy was not pressed.

It is evident that the diagnosis in this instance is as yet unproved. That it is a benign tumour seems reasonably assured in view of the absence of symptoms and a failure to grow over a span of many months, while the position of the tumour in the chest and the sharply defined contour of the mass seen in the x-ray picture suggest, in conjunction with other proved examples that have been observed or studied in the literature, a teratoma of the dermoid type.

CASE 5.—W. D., a man, aged 27 years, was admitted to the London Chest Hospital under the care of Dr. H. V. Morlock for investigation and treatment.

There had been no history pointing to chest disease until three years before, when he had an attack of pneumonia that left him with a residual cough culminating in an haemoptysis six months before admission. The blood was moderate in amount and dark in colour. The cough became productive of an offensive, purulent sputum, particularly in the morning. The sputum was negative for tubercle bacilli. Four months previously he had become more seriously ill. The cough and sputum increased and caused him to vomit in the morning and he lost about two stones in weight.

At the time of his admission to the London Chest Hospital he was very ill with an irregular temperature up to 101° F., dyspnoea, and clubbing of the fingers. Physical examination disclosed signs in the left side of the chest consisting of restricted movement and impairment of percussion note from the clavicle downwards. Breath sounds were weak in front and absent at the left base posteriorly.

The sputum, which amounted to between three ounces and five ounces daily, was purulent and contained a variety of organisms including spirochaetes. In it were seen, also, numerous yellow granules that microscopic investigation showed to consist of clumps of acid-fast bacilli. This led to the sputum being reported first as positive for tubercle bacilli, but a more detailed study showed that these organisms were acid-fast but not alcohol-fast and if treated first with alcohol they lost there acid-fast properties.

At bronchoscopy a tumour was seen blocking the lumen of the left main bronchus and pus was pent up behind the obstruction. A portion of the tumour was removed for microscopy and the section disclosed stratified epithelium from which processes were identified, dipping deeply down into the fibrocellular tissue beneath. The tissue was infiltrated with leucocytes.

A few days later the patient coughed up a portion of the tumour the size of a hazelnut and resembling a fibroma in appearance. This was followed by a severe haemorrhage and death ensued.

At autopsy the left pleural space was obliterated and the right sac contained some free fluid. A large cyst lay in the anterior mediastinum and extended into the left thoracic cavity. It was attached to the pericardium. In places the wall was composed of dense tissue three-fourths inch in thickness, while in other parts it was thinner. From the inside of the cyst were numerous papillomatous processes projecting into the cavity and some of these supported smaller papillomata. To these processes hairs were attached and the lining membrane was pitted, strongly resembling skin. A large quantity of sebaceous matter was present in the cyst.

The left lung was the seat of pneumonia with bronchiectasis at the base, while a terminal bronchopneumonia was present in the right lung. A communication was found between the cyst and the bronchial tree.

This case illustrates how an infected dermoid cyst may terminate fatally in a few months if surgical treatment is withheld in the early stages, and provides an example of pulmonary suppuration and bronchiectasis occurring secondary to pressure on the bronchus by the tumour and the aspiration of the infected contents of the cyst. It is also interesting from the bacteriological point of view, for no other case in the literature can be found in which examination of the sputum has revealed similar organisms to the acid-fast, nonalcohol-fast bacilli isolated in this instance.

Case 6.—B. S., a little girl aged 1 year and 4 months, was never seen by a doctor until after her death. From the mother's statement there was little indication of respiratory disease and no suggestion of a recent failure of general health. There was, however, a history of "mild bronchitis" for a few weeks. Suddenly, early one morning, she had a convulsive seizure and died twenty minutes later before medical aid could reach her.

At autopsy a tumour of moderate size was found in the anterior mediastinum. It was mainly solid but contained numerous cystic spaces filled with grumous material and, in some areas, haemorrhagic exudate. The tumour surrounded the trachea and great vessels to all of which it was firmly attached while, a little lower down, it was firmly adherent to the pericardium. Dissection failed to free the tumour completely from its neighbouring mediastinal structures. No other abnormality was disclosed by the post-mortem examination and no secondary deposits were found.

Microscopic study was made from three sections cut from different portions of the tumour:

Section 1: A large part of the tissue in the first section was composed of apparently normal thymus gland, including Hassall's corpuscles, separated from the main mass of the tumour by a distinct barrier of fibrous tissue.

The tissue itself presented a glandular appearance with the cells irregularly arranged but with an attempt at acinous formation. It could best be described as adenocarcinomatous.

Section 2: The second section was composed predominantly of cystic spaces lined by cubical and high columnar epithelium. For the most part the epithelium constituted a single layer, but in a few places it was heaped up and ciliated. Between these two extremes a transitional stage could be made out indicating that the one was only a variant of the other. Many of these cells were goblet cells and obviously mucus-secreting.

An interesting feature of this section was the presence of undoubted salivary gland tissue, while, in addition, pigment could be seen which failed to take the stain for free iron, thereby suggesting that its origin was not haemorrhage but more likely melanin from the choroid.

Section 3: One plaque of cartilage was seen in this section and was found nowhere else. Cystic spaces similar to the foregoing were found and the adenocarcinomatous tissue, necrotic and haemorrhagic in parts, was present in abundance.

On this histological evidence there seems little doubt that the tumour was a mainly solid, partially cystic, teratoma that had undergone malignant, adenocarcinomatous degeneration and so brought about the death of the child.

PATHOLOGY

Although the origin of teratomata of the mediastinum is believed to result from faulty development occurring in embryonic life, it is well recognised that the majority lie dormant within the chest for many years and only become obtrusive when adolescence or early adult life is reached. This is borne out by a study of 174 cases in the literature in which is given the age at diagnosis. These cases can be grouped into decades as shown in Table I.

TABLE I

AGE (YEARS)	NO. OF CASES
0- 9	17
10-19	27
20-29	68
30-39	37
40-49	11
50-59	13
over 60	1
Total	174

The specimens found accidentally at autopsy on patients who died of an independent disease have not been included. There is no predilection for sex: out of 177 instances in which the sex is stated, 91 were found in females and 86 in males.

The position of these tumours in the chest is the anterior mediastinum, but they are generally found to encroach upon one or other side of the thorax. Even when they seem to have no apparent connexion with the mediastinum, a pedicle will sometimes be discovered stretching from the tumour to the centre of the chest. Neither side is favoured in this encroachment, for in 166 cases in which information is available, 88 abutted towards the right lung field, 65 inclined towards the left side, while 13 are given as central in position.

Pathologically the tumours exhibit the utmost variation and although it is possible to recognise two definite types—the simple dermoid cyst and the more complex teratoma—this division is not a rigid one. An apparently simple cyst will be found to contain tissue that unquestionably stamps it as a teratoma, while in other instances the main bulk of the tumour is simple and cystic, but at one pole is found a solid collection of tissue containing representation from all three germinal layers. It is convenient, therefore, to describe all these tumours as teratomata and to qualify the term according to the type of tumour. Thus, for purposes of description, they will be divided into teratomata of the dermoid type and the solid teratomata.

Teratoma of the Dermoid Type.—The dermoid is the simplest and the commonest type of teratoma found in the anterior mediastinum. It is generally composed of one single cystic cavity containing sebaceous material, but it may be bilocular or multilocular. In some cases accessory and smaller dermoids are found clustering around the parent cyst but having no connexion with the main tumour. Examples of this have been recorded by Carpenter (1906) and Harris (1919) and in the first case described in this communication a small daughter cyst, the size of a hazelnut, was attached to the heart's apex. It was filled with the same yellow sebaceous fluid as the main cyst but was quite distinct from it.

Macroscopically, the dermoid cyst may remain small and latent throughout life. On the other hand, it may attain a very large size, this tendency to grow usually becoming manifest in adolescence or early adult life and seems often to be provoked by an independent respiratory infection such as pneumonia or an attack of so-called influenza. The wall of the cyst on its outer surface is normally smooth and enclosed in a fibrous capsule, but it may be bossed. It is sometimes attached to the mediastinum or sternum by a pedicle of variable length and thickness that has to be ligated and divided if surgical treatment is to be successful. Apart from this pedicle it is common to find the cyst adherent to surrounding intrathoracic structures such as the pericardium covering the heart and great vessels, the lungs and bronchi, the chest wall, sternum, mediastinum, and parietal pleura in approximately that order of frequency. These adhesions range from thin, tentative attachments that can easily be broken down by blunt dissection to a firm and intimate union requiring the knife or scissors for their release if, indeed, they can with safety be severed. From the inner lining of the cyst numerous fingerlike protuberances are often discovered jutting into the cavity, or a hard ridge is detected running the length of the tumour. This ridge may be partially made up of cartilage or bone, in some cases conforming to a well-known structure in the body such as the superior maxilla (Gordon, 1827). As a rule it is from these papillomatous processes or from this ridge that wisps or clumps of hair spring.

Microscopically, a dermoid cyst is a benign and relatively simple tumour although rarely it is found to have undergone malignant change. In the main the structure closely resembles that of skin, with an epithelial lining frequently of the squamous type, sebaceous and sweat glands present in large numbers, and hair follicles evident, together with smooth and striated muscle. Deposits of calcium and plaques of bone, cartilage, and teeth have been encountered on many occasions and a feature of many specimens is glial and nerve tissue and retinal pigment derived from the choroid (Whittemore, 1923 and Doran & Lester, 1939).

The epithelium of a dermoid cyst provides by its diversity a characteristic feature. It is not always of the stratified, squamous type but may be transitional in character, cubical and high columnar, with or without cilia. In some examples all kinds of epithelium have been present. Sometimes tissue derived from germinal layers other than ectoderm is apparent, such as salivary gland (Goebel and Ossig, 1912; Körner, 1918; and Kleinschmidt, 1920), pancreas with islets cells (Gordon, 1930; and Hablützel, 1933), thymus, thyroid, and intestine. It is this type of histological finding that bridges the gap between the dermoid cyst and the solid teratoma.

Solid Teratoma.—The solid form displays a difference from the dermoid cyst just described partly in its macroscopic and histological structure and partly in its potentiality for malignant change. A study of the tumours of this class that proved to be malignant shows that 70 per cent have arisen in the solid teratomata and 30 per cent in the teratomata of the dermoid type. The term solid teratoma is not an exact one, as many examples placed in this category are only partially solid and contain, in addition, multiple small cystic spaces filled with mucus and often containing blood. The naked eye appearance varies from a tumour that is difficult to distinguish from a teratoma of the dermoid type to a complex growth with an organised structure that deserves the name parasitic foetus (Harrington, 1932).

Microscopically, too, the tissue that comprises the more solid forms of teratomata, although derived from all of the three germinal layers, exhibits a variable complexity that may be very simple or may reveal tissue representative of every organ of the body.

PATHOGENESIS

The mediastinal terato-dermoid tumour provides a perplexing aetiological problem for which there is even yet no satisfactory explanation.

In the earlier writings there is apparent a tendency to regard the dermoid cyst as a separate entity from the teratoma and Lebert (1858), who first suggested the division, looked upon the two types as distinct. It is very tempting to adopt this view and to postulate a separate aetiology for these two groups which apparently differ so widely in their pathologic features and malignant propensities. On this basis the dermoid cyst would be classed as an inclusion phenomenon occurring in a line of natural cleavage and, thus, would fall into line with cysts of similar structure found in other parts of the body. The evidence supporting this hypothesis can be summarised as follows:

1. The structure of the dermoid cyst, studied by previous workers, often conforms closely to that of the skin and its appendages and no embryonic layer other than the ectoderm is represented. The teratoma, on the other hand, is made up of all three layers of the blastoderm.

2. The situation of the cyst in the anterior mediastinum behind the sternum is explained, although no account is taken of the rigid disposition of the tumor high up in the superior mediastinum and only exceptionally lower down at the level of the diaphragm.

3. Presternal dermoids are well recognised and many of these are found to have no connexion with the mediastinum at all (Clutton, 1887; Bird, 1904; Selby, 1906; Morestin, 1912; Cavalcanti, 1913; MacEwen, 1913; and others). In others it is the presternal dermoid that first attracts attention and at operation for its cure a communication is found piercing the sternum and opening into a second, often a larger, cyst in the mediastinum (Bergman, 1898; Hathaway, 1923). This could be named the "dumb-bell" type of dermoid tumour. Again, there have been cases recorded in which there was no demonstrable presternal cyst but an abnormality of the sternum has been noticed at an operation planned to remove a mediastinal dermoid tumour. Thus, Robertson and Brown (1929) found the sternum bifid, while Körner (1918) saw an opening in the sternum, pencil-sized, against which the tumour was closely apposed. Finally, an intrathoracic cyst is sometimes found firmly attached to the undersurface of the sternum by a distinct pedicle that could be regarded as the homologue or replica of the more perfectly developed canalised "waist" of the dumb-bell tumour. Thus, there is every gradation between a presternal dermoid with no intrathoracic association and a mediastinal cyst isolated from the surface of the body by a normally developed thoracic cage.

Attractive as this dual theory of origin might appear, the evidence adduced in support of a separate aetiology for the dermoid cyst is not irrefutable. The incidence of developmental curiosities of the sternum is small and the number of occasions on which a pedicle has been seen attaching the cyst to the sternum is few when compared with the total number of mediastinal dermoid cysts placed on record, the overwhelming majority of which are entirely intrathoracic and many of them not even adherent to the undersurface of the breast bone.

Moreover, as knowledge of the pathology of these tumours accumulated, many borderline cases were recognised until, finally, it became clear that no simple classification of these tumours into the dermoid cyst, comprising ectodermal structures only, on the one hand, and the teratoma, composed of all three layers of the blastoderm, on the other, was possible. There is little doubt that pure ectodermal cysts, resembling epidermis, do exist but it is possible that had many of the examples so described been submitted to more searching microscopic study, entodermal and mesodermal elements might have been discovered. It was the full realisation of the gradation from simple to complex that led Harrington (1937) to suggest that the terminology should be based on histology, while others workers have coined the name "teratodermoid" to include all forms.

To lay on one side the theory of a dual aetiology and to group all these tumours together postulating a common mode of origin for them serves only to clear the ground and to direct attention to a possible developmental anomaly that takes place in intrauterine life. Several hypotheses have been advanced. Collenberg (1869) suggested that the simple cysts were derived from the ectoderm of the branchial arches and were thus related to the thyroid gland. He was no doubt influenced by the specimen that he was studying, which contained thyroid vesicles, was connected to the gland by a narrow band of tissue, and was supplied with blood by the inferior thyroid artery. Marchand (1883), on the other hand, when discussing a case that was intimately connected with thymus, saw in ectodermal rests in that gland a possible source of the tumour. Other workers have demonstrated thyroid and thymus tissue in their preparations, but it is the exception and not the rule and when observed can be looked upon as a chance outcome of totipotential development. It is unlikely that a teratoma, so complex and primitive in structure and so diverse in form, can be dependent, developmentally, upon any single mature organ of the body, and its origin must be traced to an earlier and less differentiated stage of embryonic life. The "absorbed twin" theory, emanating originally from Dangschat (1903), whereby one "twin" (the autosite) engulfs the other (the parasite), takes cognisance of this and makes the presence of chorionepitheliomatous elements easy to understand. A fanciful variant of this view is the stimulation by a physiochemical process of the segmenting ovum at the two-cell stage leading to isolation of blastomeres that undergo cleavage. Two identical embryos are produced, one of which becomes parasitic upon inclusion in the midline of the other during the closure of the thorax. Other possible modes of origin are believed to be an extruded polar body or an aberrant sex cell. The latter are found in various positions in the embryo apart from the sex glands and are considered to be capable of parthenogenetic development.

The most reasonable proposition so far propounded is based upon the suggestion of Collenberg that it is in an abnormality of the third and fourth branchial arches that a likely explanation for the origin of these tumours is to be found. It is believed that the cells destined to multiply into the teratoma become separated off at this point and are carried into the thorax by the normal descent of the heart and great vessels. Such a theory accords well with the constant position of these tumours in the anterior mediastinum from the thoracic inlet to the front of the heart. Moreover, it is in the neighbourhood of the third and fourth branchial arches that development assumes its greatest degree of complexity, and ectoderm and entoderm come into a most intimate relationship with each other. This applies particularly to the epithelium, for epithelial elements of all kinds and in profusion are a striking feature of the teratodermoid tumour. Such complexity offers every inducement to misplacement of cells. Finally, it provides a possible reason for the frequency with which these tumours are found adherent to the pericardium and main arterial trunks, a fusion that is sometimes so complete as to defy all attempts at separation.

It must be admitted, however, that each theory has its shortcomings, none fits all the facts, and the final elucidation of this problem must be awaited.

CLINICAL FEATURES

The developmental nature of this group of tumours, taken in conjunction with the customary age at which the benign varieties first make their presence felt, presupposes that many, if not most, of them remain clinically latent for a number of years. This presumption is justified by Case 5 and has, in fact, been recognised for as long as these tumours have been known, for Gordon (1827) ends his original description with these words: "... I should consider the tumour had co-existence with the subject from which it was taken, and was carried without inconvenience, till inflammation in its neighbourhood excited that fluid secretion upon which its enlargement and consequent inconvenience depended." For purposes of presentation, therefore, the clinical manifestations can be conveniently grouped according to the ways in which these tumours eventually become obtrusive.

1. *Growth and the Effects of Pressure.*—A progressive enlargement of the tumour, such as has been noticed to occur most commonly in the third and fourth decades of life, will ultimately produce symptoms due

to the pressure of the mass upon neighbouring structures within the thorax.

Cough, with or without expectoration, is the commonest individual symptom. In type and intensity it displays the utmost variation which is apparent not only when one case is compared with another, but in the evolution of the disease in any one patient.

In the beginning it is short, dry, and irritating and a "barking" quality may be imparted to it and was, indeed, a striking feature of Case 1 of this series. Coughing may be practically constant but more often it is paroxysmal and the spasms, which have been known to last for several minutes at a stretch, are associated with dyspnoea and are sometimes ushered in by attacks of breathlessness (Ekehorn, 1898). The published records contain many instances in which the position of the patient has a noticeable effect upon the cough, the adoption of a certain posture, such as lying on the back or on one or the other side, serving to excite the paroxysm which is relieved as soon as the patient sits erect. Although there is often no close agreement between the situation of the tumour and the position the patient adopts, yet a study of this symptom does suggest that cough and breathlessness are both liable to be provoked by lying flat on the back or upon the side into which the mass projects. This was certainly true of the patient in Case 1, who was always to be found sitting up in bed knowing quite well that were she to lie down breathlessness and a "tickling in the throat" would inevitably follow. Although at this stage the cough is dry and useless it may produce a scanty mucoid expectoration occasionally stained with blood. A streaky haemoptysis, explicable as arising from localised congestion, may be a terminal event of every coughing fit (Bergman, 1898). This dry, hacking cough, so common an early symptom as to be almost universal, may yet be absent throughout the patient's entire illness (Phemister and associates, 1936).

Dyspnoea, with cough, is a complaint more commonly found than any other and is generally first apparent on effort. It may, however, be quite unrelated to exertion and be seen in the form of suffocative attacks that have been described by patients as "smothering spells" or "choking fits." Cordes (1859) mentions the occurrence of periodic asthma, while attacks of urgent dyspnoea with cyanosis have been observed as terminal events. The relationship that dyspnoea bears to posture has already been referred to and careful questioning of a patient, with this in mind, is often helpful in making a diagnosis.

Pain in the chest is frequently encountered in the history of these patients and resembles the pain of pleurisy in so far as it is sharp and lancinating and aggravated by coughing, laughing, deep inspiration, and effort. Pain of this kind is very likely pleural in origin and may be initiated by distension of the cyst stretching the pleural covering, by a drag upon adhesions attaching the tumour to the parietal pleura, or by an infection of the pleural envelope itself by contiguity or rupture of the cyst.

Pain, however, need not be confined to the chest wall nor need it be pleural in type. In some patients it has been felt in the shoulder with a radiation down the arm, generally along the ulnar side, to cause a feeling of pain or numbness in the hand and fingers. This manifestation was accompanied, in some instances, by paresis of the limb and muscular wasting. Even more remarkable was Tuffier's case (1919). This patient was a man who suffered from attacks of angina pectoris, the cause of which was diagnosed by Vaquez as a mediastinal cyst. At operation a large dermoid cyst was found so closely adherent to the heart and great vessels that the aorta, auricle, and ventricle could be seen beating within it and no pericardium was apparent. The "anginal" pain was completely relieved by a drainage operation and the subsequent piecemeal crushing of the calcareous wall of the tumour. Abdominal pain, as a prominent symptom, has been recorded, and in two cases (Pflanz, 1896, and Harrington, 1933) pain referred to the ear of the same side of the body as the tumour has been described.

Engorgement of the veins of the neck is not uncommonly found in conjunction with swelling of the face, and one jugular vein is sometimes seen standing out more prominently than its fellow on the opposite side. If venous obstruction is of a high grade, oedema of the feet, ascites, and anasarca become evident.

Palpitation of the heart is by no means an infrequent symptom and was one of the earliest noticed by the patient in the first case of this series. It is the result either of pressure upon the heart or of displacement of the organ by the tumour. The rhythm is often found to be irregular. Sauerbruch has drawn attention to the occurrence of paroxysmal tachycardia due to the pressure of mediastinal tumours, a symptom that may also be excited by a tampon used after mediastinal operations and relieved as soon as the plug is removed. In Case 1 the dermoid cyst was so closely adherent to the front of the pericardium that it was impossible at autopsy to separate the two structures, and such close cohesion may help to explain the prominence and early appearance of palpitations. A similar instance of firm union between the tumour and the pericardium is given by Mouat (1909). He experienced the same difficulty at autopsy when he attempted to separate the tumour from the heart and was unable to recognise the pericardium. Tachycardia is important in assessing the risk of major surgery implying, as it does, a long sustained interruption of the normal pace and sometimes rhythm of the heart action.

Pupillary changes have been mentioned in some accounts, but only rarely. A complete Horner's syndrome on the right side due to sympathetic paralysis has been seen in a patient with a dermoid cyst by Heuer (1929).

Alteration of the voice due to pressure on the recurrent laryngeal nerve has been observed. It is more commonly found with tumours that abut towards the left side than towards the right. The voice assumes a husky quality and the change of pitch is usually towards a lower tone. Laryngeal examination may reveal a paralysis of the corresponding vocal cord.

Among the more unusual manifestations of pressure are dysphagia which was encountered by Türk (1903) and, perhaps, pressure upon the vagus nerve can be invoked to explain the case of sudden death that Joël (1890) recounts in a boy of 14 years, about whose previous health nothing was known, who jumped into an omnibus just as it was starting and fell dead. The tumour at autopsy proved to be a teratoma that lay within the pericardium in front of the heart, firmly attached to, but not communicating with, the pulmonary artery.

2. *The Onset of Infection Within a Cyst.*—The advent of infection within a cyst is frequently so dramatic in onset and so grave in outcome that it may be reckoned one of the most dreaded complications of an apparently benign, symptomless dermoid tumor. It is associated, as a rule, with rupture of the cyst wall into a bronchus or pleural cavity and is liable to follow the formation of a fistula. Such an accident occasionally precedes a rapidly fatal result as in the case recorded by Christian (1902) of a woman, aged 38 years, who gave an indefinite chest history for six years, latterly with small haemoptyses and the occasional appearance of hairs in the phlegm. Quite suddenly she spat up a large quantity of blood and pus and died shortly afterward. A similar example, although not so severe, in which a fatal outcome was averted by timely surgical intervention, is recorded by Heuer in 1929. The patient, a coloured labourer, aged 53 years, experienced, during effort, the sudden onset of an acute pain in the right side of the chest together with shortness of breath and cough. The symptoms suggested a spontaneous pneumothorax but an x-ray picture disclosed a cyst with an almost completely calcified wall. It contained air and showed a fluid level, while at one corner of the calcareous envelope a crack was visible at which point a communication with the bronchial tree had become established.

Infection of the contents, however, may occur independently of fistula formation and the accumulation of inflammatory fluid is one possible explanation for that sudden increase in size that these tumours undergo. Moreover, the process may occur silently and unobtrusively as the frequency with which this class of tumour is found adherent to neighbouring structures such as lung and pericardium testifies. Heuer (1929) recounts an experience that has a bearing on this. He removed, without opening the pleura, a dermoid cyst from the right side of the mediastinum of a woman, aged 33 years. The entire tumour was removed but was ruptured during the operation and, although the contents were evacuated with a gall bladder scoop, a suppurative mediastinitis and empyema of the right side developed. There seemed no just grounds for blaming faulty surgical technique for this complication and Heuer believes that infection can occur at the same time as an acute respiratory illness and persist after the lung inflammation has subsided. The spread of infection from lung to cyst by contiguity is possible but equally possible is the transport of organisms from outlying foci into the tumour by the blood stream.

The end result of bronchial communication and infection is fever often of a hectic type; the expectoration of a large quantity of offensive sputum; haemoptysis, more copious than hitherto and which may be so profuse as to lead to a fatal issue (Ogle, 1897); clubbing of the fingers; cachexia; and death. It is not surprising, therefore, that in the early days the condition should have been misdiagnosed during life and labelled bronchiectasis, empyema, or pulmonary tuberculosis.

3. *The Formation of a Bronchial Fistula Without Obvious Infection.*—In the same way as the cyst contents can become infected without necessarily leading to a bronchial fistula, so a communication between the cyst cavity and the bronchus can become established in the absence of overt infection. That is to say, symptoms may be minimal and easily explained by the effect of pressure and yet careful questioning will reveal the presence in the sputum of "atheromatous matter," "clay-like particles," and hairs that can only have come from the contents of a dermoid cyst, and lead, when recognised, to a confident diagnosis. The subject of sputum examination, however, will be dealt with fully in the section devoted to special investigations.

4. *Presentation of the Tumour in the Neck or Chest.*—It has been possible to collect from the literature twelve instances in which the first clue to the presence of intrathoracic dermoid cyst was furnished by the appearance in the neck of a visible swelling, and in at least two other cases (Gordon, 1827; Poehn, 1871) such a tumour became obvious later in the course of the disease when other manifestations had brought the patient under observation. Soft and fluctuating, sometimes exhibiting pulsation, these tumours, although readily recognised by both doctor and patient, are not always appreciated as the outward sign of an inward and more serious disease. This was particularly evident, and quite understandable, in the days before radiography was employed. Thus, Kückmann (1874) records a soft fluctuating swelling that appeared in the left side of the neck and pushed the sternal end of the clavicle forwards, but it was some months before the diagnosis was finally established, while Koenig (1889) describes the incision of a dermoid cyst presenting in the suprasternal fossa and later trephined the sternum to evacuate the contents. It is remarkable what a long interval can elapse between the advent of a noticeable tumour in the neck and the manifestations of more serious trouble, for Shostak (1904) quotes a

young woman who remembered that she had had a tumour in the neighbourhood of the right jugular vein since childhood which showed no inclination to grow larger until after a chill contracted when she was 22 years of age. This story suggests that infection may have had some influence upon the course of the disease by leading to a sudden enlargement of the cyst. Trauma is another possible predisposing factor, for Jones (1880) related how a boy aged nine years received a blow on the sternum and three weeks later developed a tumour containing milky fluid in the lower part of the neck.

Although the neck is commonly the site at which an intrathoracic dermoid cyst presents when it approaches the surface, this is not invariable and the contents of a cyst, particularly if infected, may, like an empyema necessitatis, appear in other parts of the chest. Mandlebaum, for instance, in 1900, described a swelling in the front of the chest above the right breast at the level of the third rib and von Török, in the same year, recorded the case of a small child who had an abscess in the back three years before the existence of an intrathoracic tumour was suspected. In a separate group should be classed the partially presternal, dumb-bell type of mediastinal cyst.

5. *A Change to Malignancy and the Appearance of Metastases.*—That the onset of malignancy can mark the first departure from good health in one who harbours a benign, symptomless mediastinal tumour is evident from a survey of the published case reports. Thus, in Pinders' first case (1887), autopsy revealed a cyst, with a solid portion that was shown to be malignant, and secondary deposits in the lungs and mediastinal glands. Ritchie, also, in 1903, published an account of a man, aged 24 years, who gave a short history of only three months and whose condition was diagnosed as malignant tumour of the mediastinum with metastases in the lungs. A post-mortem examination disclosed a dermoid tumour with a solid upper pole containing many haemorrhages. Microscopy showed that the solid, haemorrhagic portion was composed of elements resembling a chorionepithelioma. Similar examples were put on record by von Eiselsberg (1903) and Ceelen (1912). The sudden appearance of metastases arising from a tumour that was either symptomless or else looked upon as benign has a bearing on the question of treatment and will be referred to again in that connexion.

PHYSICAL EXAMINATION

Apart from the appearance of the sputum and certain peculiar characteristics that it possesses there is no one physical sign or group of signs that can be regarded as pathognomonic of terato-dermoid tumours of the mediastinum. There may, indeed, be no abnormality detectable upon examination until the thorax is illuminated by x-rays. On the other hand, if pressure symptoms are evident, corresponding signs may be detected which in degree may vary from a disinclination to lie flat to urgent orthopnoea, venous engorgement of face, chest, and arms with oedema and cyanosis, pupillary changes, and disordered sweating of one-half of the face. In view of the customary position of the tumour in the superior mediastinum such abnormal signs as do exist will be elicited in the early stages in the front of the chest.

The affected side may display a noticeable bulging and Harrington (1933) tells of a young man whose tailor was the first to draw attention to an asymmetry of the chest wall five years before symptoms became added. Apart, however, from a homogeneous unilateral fullness of the thorax and the smaller and more circumscribed swellings in the neck and chest to which reference has already been made, Case 1 illustrates how a localised area of pulsation can be a striking, if albeit a misleading, feature. Movement is usually found restricted on the side of the chest into which the major portion of the mass projects.

Palpation will help to confirm a localised fullness of the thorax and any limitation of movement; it will also enable a more detailed investigation of pulsation to be made and the position of the heart's apex beat to be accurately fixed. The impulse is often displaced by anteriorly placed mediastinal tumours.

The resonance of the percussion note is impaired over a tumour of even moderate size and such an area of dullness can often be mapped on the front of the chest although the lower limit of this impairment is sometimes difficult to dissemble from the cardiac dullness with which it merges.

The breath sounds and the conduction of the spoken voice are faint or absent over the tumour. With the stethoscope, too, cardiac bruits, at the apex or at the base of the heart, can sometimes be detected and their correct interpretation is difficult.

The physical signs so far enumerated are largely referable to the mass itself; in addition, there must be recognised those signs dependent upon secondary changes in the lungs such as atelectasis and suppuration. Attendant collapse of lung can, upon occasion, simulate a pleural effusion and when a collection of serous fluid, or even an empyema, overlies the tumour the recognition of the primary cause is no easy matter. The advent of infection of the contents of a cyst with a bronchial fistula and possibly secondary suppurative changes in the lung brings as dramatic a change to the physical signs as it does to the presenting symptoms. A study of past descriptions of this malady, read in the light of modern knowledge, excites little wonder that our predecessors in the middle of the last century, compelled as they were to rely more upon an expert use of the natural senses than upon supplementary aids to implement the diagnosis, were often beguiled into error.

SPECIAL INVESTIGATIONS

Examination of Sputum.—A discussion of the characteristics of the sputum in this disease has purposely been postponed until now, in order that it may be dealt with as a whole. Careful interrogation of the patient and skillful examination of the sputum will occasionally bring to light the only diagnostic symptom and the only pathognomonic sign of an intrathoracic dermoid cyst—the expectoration of hair and sebaceous matter.

It has been possible to collect from the literature twenty-three instances where specific mention is made of the presence of hair in the sputum. The first recorded case is that of Muntz (1839), in a woman of 28 years, who stated that she had spat up hairs in the phlegm after forceful coughing ever since the age of 15 years.

In the early years before the existence of these tumours became generally recognised, the complaint of coughing up hairs was viewed with scepticism by those in attendance. Thus, Godlee, in 1889, relates how this symptom was received with amusement by the patient's husband and with incredulity by the doctor in charge, both of whom came to the conclusion that the source of the hair was a shawl that the woman was accustomed to wear. It was not until Godlee resected a rib with intent to drain an "empyema" and evacuated hairs with his exploring finger that the patient's observation was at last believed and correctly interpreted. Similarly, in Cloetta's case (1861) the expectoration of hairs in quantity was regarded by the doctor as a deception. Even nearer to our own time the significance of this symptom has not always been appreciated for Becker, in 1927, described how a woman always produced hairs whenever she had an haemoptysis and yet the true diagnosis remained unsuspected until an artificial pneumothorax had been induced and a thoracoscopy performed; while Eloesser (1933) relates how the persistent disregard of the husband's statement that hairs were seen at intervals in the phlegm led to the retention of a woman in a sanatorium for two years with presumed pulmonary tuberculosis. Nevertheless, earlier observers were not slow to realise the importance of this symptom for it was upon this observation that Nobiling, in 1869, based the first correct diagnosis of the disease made before death. Chronologically, Nobiling's case was only the third to be recorded in which this phenomenon had been witnessed.

The hairs are often expectorated singly and appear at intervals spread over a long period before more distressing symptoms supervene. As trichoptysis implies a cyst-bronchial communication, it is evident that such a communication can exist for several years without serious infection of the contents taking place. This phenomenon is not unique and has a parallel in certain cases of tuberculous pyopneumothorax in which a bronchopleural fistula can be demonstrated and yet the pus remains repeatedly sterile on culture for secondary organisms. A statement is frequently seen in the published accounts to the effect that hairs are voided only on forceful coughing. This suggests the operation of a valve mechanism that will permit the escape of cyst contents when the act of coughing is vigorous but acts as a barrier to the passage of air and infection in the opposite direction. An alternative explanation is that the greasy, sebaceous contents of a cyst act as an efficient barrier to the exit of hair by blocking the opening. It is only when expiration is sudden and forceful that this plug is dislodged. It is by no means rare, however, to notice that the hairs in the sputum mug are matted together or attached to concretions composed of fatty and atheromatous matter. Frogman exhibited such concrements before the Society of Swedish Physicians (Ekehorn, 1898) and Heuer (1929) records the expectoration of "claylike particles" with hairs attached. The colour of the strands bears no relationship to the natural colour of the hair of the host, the hue being as a rule nondescript, occasionally blonde, and in one instance reddish.

In other respects the examination of the sputum supplies valuable additional evidence of a dermoid cyst. Sebaceous matter, with its oily, greasy consistency, is characteristic and fat droplets may be seen. More solid elements are sometimes forthcoming, which have been described as atheromatous matter and claylike particles. Cholesterin crystals are distinctive but not pathognomonic and can be recognised by the glistening, opalescent sheen that they impart to the surface of the fluid. Finally, reference must be made to the observation by Foucher (1901) of small, yellow, round balls resembling actinomycotic granules that occurred in a sample of sputum, and similar bodies were noticed in the fluid removed during the exploration of a cyst by Fanano (1937).

Exploration of the Cyst or of a Presenting Swelling.—The contents of a cyst have been tapped on numerous occasions in the mistaken belief that the diagnosis was a pleural effusion, and in some cases the disease has remained unrecognised until a rib has been resected for an "empyema thoracis." In either event the character of the fluid obtained was sufficient to clear up the diagnosis in most instances. It must be remembered, however, when a formal exploration of a cyst is contemplated, that unless a wide bored needle is used, a "dry tap" is likely to result owing to the thickness of the fluid that is sometimes encountered.

Although exploratory puncture has been safely and successfully accomplished and information of value obtained from the procedure it is not to be lightheartedly recommended. To drive a needle of wide bore across what may be a free pleural space into a cyst that is very likely under tension is fraught with the danger of the escape of the contents into the pleural cavity there to excite a serous effusion or an empyema. There is greater justification, however, for needling a presenting swelling of the chest wall. It can be assumed with some confidence that in that situation the two layers of the pleura will have become firmly united and the danger of defiling the pleural space is negligible. Even so, the exact position of the adherent area must be first checked by x-ray localisation. Apart from this solitary exception, diagnostic exploration should not be carried out as a routine procedure as the difficulty of determining whether the pleural space is free or otherwise is great, while if the true nature of an intrathoracic dermoid cyst is uncertain, an aneurysm of the aorta is presumably still one of the possible diagnoses.

Radiology.—As in every department of modern chest medicine, radiology has contributed in no small measure to the early and accurate recognition of intrathoracic tumours. The x-ray film of today rivals the autopsy table of yesterday in supplying the first intimation of the presence of a mediastinal dermoid cyst. Moreover, there is every likelihood of an increase in the number of symptomless tumours recognised in this way if mass radiology for the detection of early pulmonary tuberculosis is seriously applied to large groups of people or to militiamen.

Türk (1903) was the first to apply radiography to the diagnosis of an intrathoracic dermoid cyst and from that date onwards an anteroposterior picture of the chest has been usefully employed, not only in envisaging the tumour, but in separating from the main mass such complications as pleural effusion or secondary pulmonary abscess. It has only recently been realised, however, that a radiological investigation is incomplete if one picture taken in one plane only is studied. A lateral view of the chest, in conjunction with an anteroposterior picture, is necessary if a sound opinion is to be formed as to the probable nature of a mediastinal opacity.

A study of a terato-dermoid tumour in these two planes will show that it is usually circular or globular in shape with a clear-cut edge. The contour, however, may be irregular and lobulated, and Hammarskjöld (1934) attaches importance to this in distinguishing a solid teratoma, with its bossing, from the uniform and smooth margin of the dermoid cyst. Another point of the utmost diagnostic importance is the position of the shadow. Clinical examination will often suggest that it lies in front; the lateral picture confirms this impression by showing that it occupies the anterior compartment of the mediastinum immediately beneath the sternum. There are few other intrathoracic tumours that are similarly disposed. This fact is clearly brought out in the x-ray reproductions of the first four cases here described. But the value of the lateral picture is not confined to fixing the site of the tumour; in conjunction with oblique views it will sometimes demonstrate a pedicle that attaches the tumour to the mediastinum or sternum. This observation was first made by Hammarskjöld (1934). The pedicle is not always present and is not always apparent radiographically but, if it is seen, it possesses diagnostic significance and has recently been recognised and correctly interpreted by Harrington (1937) and by Smith and Mills (1938).

The main mass of the tumour, as a rule, has a homogeneous density but occasionally this uniformity is interrupted by small, more opaque areas that can be identified as teeth or plaques of bone (Singer, 1928; Lamarque and Chaptal, 1930; Bengolea, 1933; Harrington, 1933; Lambret, 1933; Harrington, 1937). This greatly assists in the diagnosis. Areas of calcification in the wall of a cyst are also opaque to x-rays and rarely the wall is wholly composed of calcareous matter resembling a shell. Difficulty may sometimes be met in differentiating bone and teeth from calcareous deposits in lung and hilar glands, but lateral or oblique skiagrams will show that the opacity lies in the wall or substance of the tumour and not outside of it.

A skiagram taken in the erect position will sometimes disclose a fluid level in the mass. This is an important observation, indicating that the major portion of the mass is cystic and that communication with a bronchus exists. If, in addition to a fluid level, the tumour can be demonstrated in the anterior mediastinum no other diagnosis than a dermoid cyst is permissible on radiological grounds. Phemister and his coworkers (1936) have recently made an interesting radiological study of a dermoid cyst of the mediastinum and have demonstrated a "fluid level" in it in the absence of air and, hence, in the absence of a cyst-bronchial communication. Their contention is that, provided the patient *sits quite still and erect for a short time before the picture is taken*, the fat in a cyst will separate off from the aqueous contents at body temperature and rise to the surface like cream, a line of demarcation forming between them. The cyst they studied was removed at operation, and an x-ray picture taken of the excised tumour again showed the "fluid level." After cooling, the fat broke up into clumps, floated in the aqueous fluid, and the phenomenon was no longer seen. A portion of the cooled contents was heated in a cylindrical vessel and brought to 37° C. and maintained thus for twenty-four hours. The fat became fluid and the line of separation on an x-ray film was obvious. It is not always possible to demonstrate Phemister's line and its existence may, to some extent, depend upon the nature of the fat. This has only been chemically analysed in a few isolated instances.

Bronchography with opaque oil does not occupy such an important place in the investigation of intrathoracic tumours as it does in other forms of nontuberculous pulmonary disease. Kerr and Warfield (1928) employed lipiodol for the first time to reveal bronchial narrowing due to a cyst but the pioneer of "contrast radiography," used in its broadest sense, was Morone (1909), who injected milk of bismuth into a sinus in the neck and an x-ray picture of the chest showed that the sinus communicated with a cavity behind the sternum. For one purpose, however, bronchography is invaluable. It is often difficult to be certain how far such symptoms as foetid expectoration or haemoptysis are attributable to an infected cyst or to secondary bronchiectatic changes. It is important that this distinction should be made if radical surgery is contemplated lest too rosy a hope of complete and lasting recovery is placed in an operation that is inevitably difficult and dangerous. A successful bronchogram will supply the answer.

The kymogram gives an interesting pictorial record of pulsation but its usefulness will remain limited until it is possible to infer more accurately than at present whether such pulsation is inherent or transmitted. A benign tumour will often display more vigorous "notching" of its edge than an aneurysm of the aorta. Likewise, the tomogram offers relatively little information of value.

Fluoroscopic screening of the patient is helpful. Minute detail cannot be expected of this method but the relationship of well-defined structures within the chest can be made out with an accuracy that a series of films will often fail to supply. Moreover, it will assist in finding the correct angle at which to take a permanent oblique record. A careful screening of the patient is indispensable in differentiating between a tumour and an aneurysm of the aorta, while a barium swallow not only outlines a portion of the aorta but demonstrates any interference there may be with the function of the oesophagus.

Thoracoscopy.—The inspection of a mediastinal tumour with a thoracoscope after a preliminary pneumothorax has been induced will occasionally supply valuable information. In Becker's case (1927), for instance, this procedure led to the correct diagnosis of a dermoid cyst after a label of bronchiectasis had been attached to the patient for two years. By this means the smooth, glistening wall of the tumour can be visualised and the nature and extent of the attachment to surrounding organs can be assessed. Moreover, with a one-cannula operating instrument of the Chandler pattern the tumour can be palpated with the blunt-ended diathermy knob and a conclusion reached as to whether it is predominantly cystic or solid. It would be wise, however, to refrain from any attempt to aspirate the contents in the presence of a free and uninfected pleural space.

Bronchoscopy.—In the investigation of a solid mediastinal tumour, bronchoscopic examination is of limited value and is confined to demonstrating a narrowing of the lumen of the tube from pressure exerted from outside. If the tumour is cystic and a fistula exists, more information from endoscopy may be expected. Thus, the characteristic contents of the cyst might be obtained for the first time by bronchoscopic aspiration, while the appearance of pus welling up from the tubes distal to the opening of the cyst should arouse suspicion of secondary bronchiectasis. In Case 5, reported in this communication, a portion of tissue removed through the bronchoscope bore a close resemblance to skin and did not suggest bronchial mucous membrane. This biopsy was strongly in favour of a tumour of the dermoid type.

The Aschheim-Zondek Test.—The Aschheim-Zondek or Friedman test deserves prominence in this discussion not so much for the diagnostic aid that it has rendered in the past as for the help it might give in the future. Up to the present it has seldom been applied to mediastinal tumours.

Zondek, in 1929, showed that men suffering from teratoma of the testis excreted prolan A in the urine and gave a positive Aschheim-Zondek test. Work was extended along the same lines by Ferguson (1934), who found that in 117 consecutive cases of teratoma testis all gave a positive result in contrast with 100 cases of benign lesions of the organ in which the result was invariably negative. He concludes that a significant excretion of prolan A in the urine (500 mouse units per litre) has never been observed in the male except in teratoma testis, extragenital teratoma, or chorionepithelioma. Furthermore, if the primary tumour of the testis is removed the reaction becomes negative within seven days of operation and the presence of metastatic deposits can be inferred from a return of a positive Aschheim-Zondek test anytime up to three months before symptoms or physical signs become evident. By putting the test on a quantitative basis, Ferguson showed that of all the types of embryonal tumours of the testis the chorionepithelioma led to the greatest excretion of prolan A.

The application of this work to mediastinal tumours will be appreciated when it is recalled that many teratomata found in the mediastinum are composed of undifferentiated tissue that in some instances has been designated chorionepitheliomatous. A positive Aschheim-Zondek test in a man with a normal testis, or in a female if pregnancy can be excluded, together with the radiological demonstration of a mediastinal opacity would go a long way toward disclosing the teratomatous, and probably malignant, nature, of the latter. Hammarskjöld (1934) found a doubtful positive Aschheim-Zondek test in a male, aged 22 years, who was operated upon for an intrathoracic tumour which was later shown to be a teratoma with malignant change, while two years previously Kantrowitz (1932) had reported a case of a 22-year-old male who was found, at autopsy, to have a teratoma of the anterior mediastinum containing chorionepitheliomatous elements with a positive Aschheim-Zondek test in the urine and in extracts of the tumour tissue. Admittedly, a necessary step in the diagnosis of a primary extragenital chorionepithelioma in the male is a careful histological examination of the testes, and it is doubtless true that in many cases so designated this examination has not been sufficiently searching. In Kantrowitz's case already quoted, many thin sections of the testicles were studied before the organs were pronounced free of tumour cells. Frank's (1932) contention, therefore, that where a circumscribed three-layered teratoma is found in the mediastinum, it can be definitely stated that the primary origin of the chorionepithelioma is at that site is upheld. Friedman's tatic changes. It is important that this distinction should be made if radical surgery is contemplated lest too rosy a hope of complete and lasting recovery is placed in an operation that is inevitably difficult and dangerous. A successful bronchogram will supply the answer.

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a tumour, proof resting upon post-mortem examination. In other accounts the clinical diagnosis of phthisis or consumption was later found to have been incorrect, the symptoms being the result of the complicating factor of infection. Today, when the sputum is examined for Koch's bacillus almost as a routine and the application of x-rays to diagnosis has become universal, such confusion has only to arise to be dispelled.

It will generally be only a matter of time before the foregoing conditions are distinguished from the class of tumour under discussion or else allotted their rightful, subordinate place in the clinical picture. The differential diagnosis between the terato-dermoid tumour of the mediastinum and other types of tumour occurring in the thorax is harder to reach. The more characteristic features of the terato-dermoid tumour have already been described and it follows that in this differentiation the greatest difficulty will be experienced in the case of those tumours that lie towards the front of the chest beneath the sternum. Tumours of the thymus gland illustrate this point. Histologically, these tumours form a complex group that has so far defied successful classification and for this reason the term *thymoma* is used to embrace all varieties. Many of these tumours are highly malignant and almost all of them are locally invasive. Among the ill-defined variations and transitional forms of this group is included the lymphosarcoma, which is fairly generally recognised, while the use made by some authors of the term teratoid type of malignant thymoma (Andrus and Foot, 1937) emphasises the structural similarity that may exist betwen thymoma and teratoma. Clinically, the comparatively short history associated with the symptoms and signs of malignancy serves to distinguish a thymoma from all but the malignant forms of the terato-dermoid class of tumour, although the position in the mediastinum occupied by both groups is identical. Moreover, the response to deep x-ray therapy is a valuable aid to diagnosis, for while lymphosarcoma and malignant thymoma are both radiosensitive the teratoma remains unchanged. Finally, use may be made of the observation that in some instances a tumour of the thymus is associated with the clinical features of myasthenia gravis (Norris, 1936).

Lipomata and *fibrolipomata* have been recorded in the mediastinum and may attain to a great size. Their presence can be inferred by means of an exploring needle, as a "dry tap" will result even if a needle of wide bore is used. No firm resistance is encountered and a sensation similar to probing a pat of butter is experienced (Walker, 1936-37).

Carcinoma of the bronchus, particularly the "paramediastinal" form with a sharply defined lateral edge, must be considered although in most cases in which the true nature of this condition is in doubt confusion arises over other causes of pulmonary atelectasis, suppurative or chronic inflammatory changes, or other affections of the mediastinal glands. Rarely, however, a carcinoma of bronchus can develop in such a way as to resemble closely a teratoma. An example of this has recently been studied at the London Chest Hospital. The patient, a married woman aged 24 years, was admitted for investigation with a provisional diagnosis, made by a radiologist, of a dermoid cyst of the mediastinum. The tumour lay in front of the chest and possessed a clear-cut posterior edge. It had invaded the chest wall to present as a swelling in the region of the second left interspace. The diagnosis of carcinoma was finally made by exploring the presenting swelling and removing a piece of tissue for biopsy. Another variety of malignant tumour that deserves mention is that first described by Pancoast, in 1932, and called the *superior pulmonary sulcus tumour.* It lies in the thoracic inlet and is characterised by pain and numbness down the arm of the same side of the body as the tumour with wasting of the small muscles of the hand, Horner's syndrome, erosion of the ribs and vertebral bodies, and a relatively small opacity on the x-ray plate at the summit of one apex. Pancoast originally believed that these tumours were embryonal in origin, but a recent study by Owen, Hewer, and Whitaker (1938) suggests that they are more probably apical bronchogenic carcinomata. In any event, their clinical manifestations are more dependent upon their position in the chest than on their essential pathology and there seems little justification for perpetuating a separate aetiology and distinctive title for these growths.

Tumours that are found almost exclusively in the posterior mediastinum are only very rarely teratomatous in origin and, hence, the position in the chest that the tumour occupies, as revealed by a lateral x-ray picture, is of the utmost importance. Admittedly, Fuller and Jagger (1927) have recorded a dermoid cyst of the back which lay in the erector spinae muscle, but there was no evidence that its site of origin was mediastinal or even intrathoracic. In view of the fact, however, that tumours situated behind do occasionally advance forward they may cause difficulty and so enter into the differential diagnosis. They include the *neurofibroma,* the *ganglioneuroma,* and the *xanthoma,* a curious, yellow-tinged, benign tumour of uncertain aetiology and great rarity that has been made the subject for study by Phillips (1938). These are often amenable to surgery. The neurofibromata and ganglioneuromata are closely allied and have been classed together as "neurinomata" (Carrière and Huriez, 1937). Histologically, those tumours exhibit an extreme degree of pleomorphism and examples are found exhibiting every gradation from well-differentiated to undifferentiated cells. With lack of differentiation goes malignancy and these are the types commonly found in children; occurring in adult life the tumour is more usually benign. Thus, Cooley and MacNamara (1940) record the case of a medical practitioner, aged 62 years, who died of pneumonia. He was known to have had a thoracic tumour for eighteen years and at autopsy this was proved to be a ganglioneuroma. These tumours are more commonly found high in the chest and in the paravertebral gutter; the capsule is sometimes incomplete and they may invade the spinal cord, while the recognition of Horner's syndrome, if this is present, is a valuable aetiological sign.

An intrathoracic *cystic lymphagioma* has been recorded. It usually arises from the posterior mediastinum and is composed of many cystic spaces containing a watery fluid. It can sometimes be surgically removed (Skinner and Hobbs, 1936).

There is one variety of mediastinal cyst, believed to be congenital and embryogenic in origin, that in the literature seems, in many cases, to have been confused with a teratoma. It is the *mediastinal cyst of gastrogenic and bronchogenic origin.* Morley Fletcher (1897) recorded the first example that it has been possible to trace, in a girl aged 6 years. He regarded the specimen as a congenital cyst arising in connection with the foetal oesophagus. Mixter and Stewart (1929) and R. E. Smith (1930) have contributed further examples. Histologically, the wall of the cyst in some specimens is indistinguishable from normal stomach, in others it resembles bronchus, while in a few both types of tissue are found. Their structure which conforms to a distinct pattern and is more stereotyped than the teratomatous cyst, their position in the posterior mediastinum, and their clinical recognition at a relatively early age form good grounds for invoking for them an aetiology different from that of the teratomata already described.

Passing reference must be made to certain well-recognised causes of *mediastinal glandular enlargement*—tuberculosis, Hodgkin's disease, gummatous infiltration, carcinomatous deposits, myeloid and lymphatic leukaemia—if such glandular masses are not to constitute a diagnostic error. Each of these conditions presents certain individual clinical features or else is amenable to specific laboratory and therapeutic tests the proper evaluation of which, combined with a radiographic demonstration that the mass is central and not anterior in position, should enable the correct diagnosis to be reached after only transient hesitation.

There are two affections of the cardiovascular system that enter into the differential diagnosis of the tumours under consideration: *aneurysm of the aorta* and *cysts of the pericardium.*

The distinction between thoracic aneurysm and tumour is not always easy to draw and in spite of modern methods of investigation there is no occasion today to disagree with Graham Steele (1911) who, in a Bradshaw lecture devoted to this subject, remarked, "All who have had much acquaintance with the two conditions—neoplasm and aneurysm within the chest—will, I think, admit the difficulty occasionally experienced in distinguishing between them, even when the greatest care is taken." That a saccular aneurysm arising from the first part of the aorta and extending forwards can so closely resemble a dermoid cyst or benign tumour of the mediastinum as to cause perplexity is evident from the rare occasions in which an exploratory thoracotomy has been undertaken. In one such instance, studied at the London Chest Hospital in 1935, it was the absence of pulsation that falsely suggested the possibility of a benign tumour. Even when the chest was opened there was no pulsation apparent to the eyes or the fingers. The x-ray pictures of this case, which are similar to those of a dermoid cyst, have been reproduced in an article by Sparks (1937-38). The clinical appreciation of pulsation of an intrathoracic mass has for many years been taken as presumptive evidence of an aneurysmal origin. Now that fluoroscopy and the kymogram have proved an advance on palpation in the detection of pulsation, and in view of the difficulty of determining by such means the inherent or transmitted nature of the pulsation observed, some revision of the significance of this sign as a point in the differential diagnosis between aneurysm and neoplasm is needed. In fact, recent clinical and fluoroscopic studies suggest that the converse of what has been traditionally believed is more often true, namely, that it is the tumour which pulsates and the aneurysmal sac which may fail to do so. Thus, Burvill-Holmes (1934), discussing this subject, attaches little importance to pulsation and remarks that, in his experience, pulsation of an aneurysmal sac is the exception rather than the rule. A possible explanation is this: a saccular aneurysm may communicate with the parent vessel through a relatively narrow ostium and its inner wall becomes lined by a layer of clot. Only a limited and sluggish circulation, therefore, is possible within the sac and this is insufficient to endow the sac with inherent pulsation. It follows that any pulsation that is apparent must be transmitted, the driving force being a diseased and enfeebled syphilitic aorta. A tumour, on the other hand, rests upon a normal, healthy vessel and has a vigorous pulsation imparted to it which the tumour, in its turn, transmits. Although in the differential diagnosis between these two conditions the presence or absence of pulsation must be carefully looked for and the result correctly interpreted, there are other points which will help in the decision. These include the fluoroscopic visualisation of the aorta, if possible throughout its whole length; the quality of the aortic second sound and the presence of aortic regurgitation; the age of the patient; and the serological tests for syphilis which are usually, but not invariably, positive in aneurysm. Such signs as tracheal tugging and a displacement outwards of the apex beat are open to misinterpretation and the possibility of syphilis and a dermoid cyst occurring together in the same patient should not be overlooked (Gallavardin and Bocca, 1921).

Of the pericardial cysts, the one most likely to cause confusion with the terato-dermoid tumour is the so-called inflammatory pericardial diverticulum which has been comprehensively studied by Kienböck (1927) and Kienböck and Weiss (1929). These cysts are believed to be encapsulated, saccular collections of fluid resulting from a previous pericarditis. They are seen, radiologically, as oval, multiangular, often pulsating, masses more often jutting out from the right heart border than the left. In the successful recognition of these diverticula Kienböck stresses the importance of remembering their existence, their stationary size over a long observation period, the detection of cardiac dilatation, and the normal appearance of the aorta.

There remains to be enumerated a number of other conditions that may be confused with terato-dermoid tumours of the mediastinum:

Solitary cyst of the lung or bronchus may temporarily give rise to doubt, especially if the cyst is filled with fluid and lies anteriorly beneath the sternum. This type of cyst, of which the "paratracheal" cyst is a variety and which probably bears a developmental affinity to those of bronchogenic origin already discussed, and the dermoid tumor under consideration have both, at times, to be differentiated from an *encysted interlobar empyema.*

Hydatid cyst of the mediastinum, although rare in this country, enters into the differential diagnosis in districts where echinococcal infection is rife. The expectoration of the characteristic sputum containing hooklets clinches the diagnosis while the complement fixation test and Casoni reaction supply useful evidence.

Retrosternal goitre, particularly if unaccompanied by thyrotoxic symptoms or signs, may so resemble a mediastinal tumour as to remain undetected until operation. The characteristic wedge-shaped opacity on the x-ray plate is not always recognisable, and the elevation on swallowing is not always detected nor is it pathognomonic of substernal goitre. The demonstration of calcium in the mass on the x-ray film, when it occurs, is an added cause of confusion.

Achalasia of the cardia with a *megaoesophagus* can also simulate a mediastinal mass until a barium swallow clears up the diagnosis (Chandler, 1939). *Spinal caries* with a cold abscess tracking forwards is another condition that must be borne in mind, for Fanano (1937) in his account of a case of a mediastinal dermoid cyst mentions how such a cold abscess had presented similar signs. Careful radiography in the lateral plane and the exploring needle should dispel all doubt.

COMPLICATIONS AND TREATMENT

The only form of treatment that will prove of any lasting avail for a teratoma of the mediastinum is surgical intervention. All measures of a medical character must be viewed as purely palliative; deep x-ray therapy and radium, which would not be expected to influence the course of a benign dermoid cyst, has been found disappointing even when applied to a malignant teratoma. Thus, Bevan (1933) inserted radium needles before advising surgery in an infected dermoid cyst without any effect upon the size of the tumour or the quantity of discharge, while

Hammarskjöld (1934) failed with deep x-rays in a teratoma that was subsequently shown to contain chorionepitheliomatous elements. Repeated aspiration of the contents of an infected dermoid cyst that is adherent to the chest wall will temporarily relieve such distressing symptoms as cough, dyspnoea, and profuse expectoration but amelioration will be short-lived.

The surgical approach to an intrathoracic tumour should never be lightheartedly recommended despite the comparative safety with which an exploratory thoracotomy can now be undertaken, for any attempt to remove the tumour entails a potential risk to the life of the patient. For this reason it is necessary, before embarking upon a discussion of the possible methods of operative treatment and their results, to form some idea of the ultimate fate of such a tumour if it is left alone and to call to mind certain complications and accidents to which these tumours are singularly liable and which, if allowed to occur and progress, so hamper the surgeon as to render his task many times more difficult than it need be if not actually impossible of execution. This applies particularly to those tumours which in the future may be first recognised by routine radiography at a stage when symptoms are absent.

There are a few instances in which the tumour has been discovered accidentally either in the course of a routine autopsy performed for an independent disease or during an anatomical dissection, and out of the records of 209 cases that have been consulted there were nine, or 4.3 per cent, in which it was clear that the tumour did not contribute in any way to the cause of death. In eight of these it was a teratoma of the dermoid type and in only one was it a more complex teratoma with multiple cystic spaces. The cause of death and the age of the patients in these cases were as follows: pneumonia, 61 years (Foà, 1874); bulbar paralysis, 53 years (Pinders' second case, 1887); tuberculous meningitis, 30 years (Marfan, 1891); pulmonary tuberculosis, 30 years (Kretz, 1893); phthisis, 37 years (Sommers, 1901); discovered during dissection (Ferra and Sice, 1910); gunshot wound in battle, 25 years (Hörnicke, 1922); pulmonary embolism following laparotomy, 65 years (Gordon's second case, 1930), and carcinoma of the stomach, 61 years (De Stella and Beerens, 1935). The average age in this list is noticeably higher than is usual in this disease and if it is assumed that the younger patients would have continued symptom-free in the absence of intercurrent disease or violence, an event which is unlikely, it would seem that only 4.3 per cent of terato-dermoid tumours fail to produce symptoms at some stage in the course of their career.

Second, a study has been made of the histories of those patients with a terato-dermoid tumour who died of this disease and who were submitted to no form of active treatment other then exploratory puncture. There were forty-seven such examples. In forty of these patients the duration of symptoms referable to the tumour is given with some exactitude while in the remainder the author has been content to employ such phrases as "sudden acute illness," "acute pneumonia," "long illness," "sudden death," "a few weeks," etc., in his description of the course of the disease. In the forty cases in which the facts are either given or can be worked out, including both the benign and malignant forms, the length of history was found to vary from fourteen years to two weeks and the average length of time elapsing between the first unequivocal symptom of the disease and the death of the patient proved to be 1.8 years. When account is taken of the diversity of course that the disease runs it is clear that this figure of 1.8 years as the average span of life of the recognised but untreated tumour has obvious limitations from the practical viewpoint. Yet it serves to emphasise the need for treatment more especially when it is recalled that it is in the late teens, twenties, and early thirties that the first symptom comes to light in most cases.

Those complications and episodes that are liable to develop and that require surgical intervention to relieve or to prevent them can be conveniently grouped under five headings:

1. *A progressive increase in size:* An increase in size commonly occurs, as has been stated, in the third and fourth decades and leads to purely pressure symptoms. If surgical treatment is contemplated it should be applied early because the attendant risks increase more in a geometrical than an arithmetical progression with an increase in the size of the tumour. Allow the tumour to double itself in size and the difficulty and danger of its complete extirpation are increased almost fourfold. Growth as a rule is not rapid and any tendency for it to occur can be recognised early by serial x-ray pictures.

2. *The spontaneous development of a cyst-bronchial communication:* The development of such a fistula can occur instantaneously and lead to sudden death following haemoptysis or to a quick decease consequent upon infection of the cyst contents or secondary pulmonary suppuration. More often the disease assumes that slow, progressive, downhill course associated with chronic intrathoracic suppuration and with an accompanying deterioration in the general health and resisting powers of the patient that the thoracic surgeon has learned to dread. A parallel is seen in the difference in the mortality figures of a lobectomy performed upon a "clean" case of bronchiectasis and a similar operation undertaken after months of infection have laid their stamp upon the patient (Romanis and Sellors, 1936). If success is to be achieved the surgeon should be allowed to forestall infection and not be asked to cure it.

3. *Rupture into adjoining structures:* The bronchus is the commonest structure so implicated and by virtue of its importance has been discussed separately (vide supra). Other instances of ruptures

have been recorded, such as those into the aorta (Büchner, 1853), pericardium (Cordes, 1859), superior vena cava (Stein, 1917), and pleural cavity (Wheeler, 1939). With the exception of the last named, surgical relief either will be sought with but a slender chance of success or will come too late.

4. *The development of adhesion binding the tumour to related structures:* Development of adhesion is, to some extent, dependent upon infection but may occur in the absence of any obvious infection of the contents of a dermoid cyst. In this latter group the mechanism of the production of such adhesions has given rise to speculation. They may possibly be the direct result of previous independent respiratory illnesses such as pneumonia and influenza, or it may be that the situation of the tumour upon a constantly beating heart gives rise to adhesions due to a continuous minor trauma associated with this pulsation. At all events it is evident that the longer a tumour is allowed to remain within the chest the greater will be the opportunity for such adhesions to develop to the embarrassment of the surgeon once the decision to operate has been reached.

5. *The change to malignancy:* When once the conversion from benignity to malignancy has taken place there is very little chance of ultimate recovery. No case can be traced in the literature in which a radical operation in such circumstances has proved permanently successful. Of the 209 reported cases in the literature that have been consulted for details, twenty-seven were malignant and this does not include Lanza's (1937) unique example of a secondary deposit from a primary laryngeal growth found in a simple dermoid cyst. This puts the incidence of malignancy at 12.9 per cent which agrees well with Jacobs' (1929) estimate of 10 per cent. Malignant transformation in a previously benign tumour can occur at any time and the change may be unaccompanied by any collateral phenomenon such as a sudden increase in the size of the tumour (Hammarskjöld, 1934), the first intimation that it has taken place being the appearance of metastases in the lungs or elsewhere, thereby precluding the possibility of curative treatment.

It is perhaps permissible, in the event of a symptomless tumour being discovered accidentally by radiography, to withhold radical treatment if any other physical disability or an unfavourable mental reaction contraindicates it, provided that serial x-ray pictures demonstrate no tendency on the part of the tumour to grow. In all other cases, bearing in mind the limitations of medical treatment and having regard to the complications already enumerated, which may quite suddenly and without warning drastically alter the clinical picture, a surgical attempt to obtain a cure should be seriously contemplated and, in most instances, advised.

As a background for modern procedures it would not be unprofitable shortly to trace, historically, the development of surgery in this field and to sketch the evolution of a technique which is still developing.

Many of the earlier operations were undertaken in the absence of an accurate diagnosis. Poehn (1871) was the first to apply surgical principles when he introduced a trocar and cannula into a dermoid cyst for the evacuation of pus. The result is not stated. Kückmann, in 1874, by a similar procedure reduced the size of the sac and achieved an improvement by closing a bronchial fistula. Butlin (1883) in Holmes and Hulke's *System of Surgery* contributes the first record in English of a drainage operation on a mediastinal dermoid cyst, but the earliest complete and detailed account in the English language is an article by Godlee in 1889. He operated upon a young woman for a supposed "empyema" and the true nature of her illness was not disclosed until a rib had been resected and the cavity explored with the finger. Numerous operations were performed upon this patient with the object of removing the papillomatous processes that lay within the cyst and reducing the size of the cavity but, as Morris (1905) relates from a personal communication he received later from Godlee, the woman died of "pyaemia" four years after the first operation. Bastianelli (1893) lays claim to being the first surgeon completely to remove, in one stage, a dermoid cyst of the mediastinum. The operation was curative. Von Eiselsberg, operating on the patient in Türk's case (1903), achieved a similar success and in the same year Madelung (1903) cured his patient by radical surgery after rib resection and drainage had left a fistula. From then on, many attempts were made either to remove a tumour completely or, failing that, to remove it partially and marsupialise the remainder to the chest wall. A varying degree of success attended these exploits, some of which were performed in multiple stages and required supplementary thoracoplastic operations for their completion.

There can be hardly a doubt that from 1918, the practical experience gained during the years of the World War of 1914 to 1918, was put to good use. Thoracic surgery, as a separate branch, was born and the surgeon learned, after mastering the problem of the open pneumothorax with the assistance of an overpressure anaesthesia, that technical accomplishments, undreamed of hitherto, had only to be practised to be made effective. In these postwar years more than half the literature of the subject has been built up and in place of partial success or improvement can now be read accounts of radical cures. American workers have been most prolific in output, but this country has not lagged behind in originality for it was in 1914 that Morriston Davies effected almost a complete cure from a many stage attempt to deal with a teratoma, while Tudor Edwards, in 1926, reported three examples of the success that can follow radical surgery. A review of the literature, with particular reference to the results of surgery, has been undertaken on a few occasions in the past, notably by Aurousseau (1924) and Howard Beye (1926).

From a study of the medical literature bearing on this subject there exists a choice of three possible procedures, leaving on one side aspiration of the contents of a cyst, a manoeuvre as deftly performed by the physician as by the surgeon and having only a palliative effect. The results of treatment along these three lines of approach in the hands of previous workers will now be considered.

1. *Rib resection and drainage:* It is apparent, from a study more especially of the earlier writings, that rib resection has been used either, mistakenly, in the treatment of an empyema or else as a last resort to lend aid to a patient in extremis. The results are bad. As the only method of treatment it has been employed on thirty-one occasions, of which thirteen patients were improved, fourteen died, and in four the result was not stated. Sometimes it has been used as a preliminary measure to more effective surgery later and, in this connexion, it serves not only a useful but a necessary purpose if the case demands it. Apart from this solitary indication, however, it has little to recommend it.

2. *Partial removal and marsupialisation:* It sometimes happens, when adhesion between the tumour and other organs is unusually firm, that partial removal is the only course open to the surgeon and is the makeshift result of an exploratory thoracotomy designed to remove the entire tumour. In fact, the operator should be prepared to accept this as a possible outcome of every exploratory thoracotomy in this disease, and should reason that it is wiser partially to remove and marsupialise a tumour successfully than to run an overwhelming risk of a major surgical disaster. Moreover, if the nature of the tumour and limited adhesions permit a radical operation but the condition of the patient causes anxiety, partial removal and marsupialisation do not preclude the possibility of a second attempt at a later date; thus, such an operation has often proved but the first step towards a multiple-stage complete removal of the tumour. When this operation is employed alone the results are slightly more gratifying than rib resection and drainage partly, no doubt, because the condition of the patient has allowed this more serious interference. Of fifteen patients so treated in the past, nine were regarded as improved, four died, while in one the result was not stated, and in one it was described as unsatisfactory. A large proportion of the tumour can be removed in this way and the dangers of increasing girth and subsequent infection largely banished from the future clinical picture. Yet the operation cannot be classed as radical in view of the theoretical possibility of malignancy supervening in that portion of the tumour that is left. There is, also, every likelihood of a persistent sinus permanently discharging secretion, although the fact that such a sinus can be made to heal has been demonstrated in Case 3.

3. *Complete excision of the tumour in one or more stages:* Complete excision is the ideal to be aimed at when surgical intervention has been decided upon, and in the hands of experienced men has given very satisfactory results. It has been attempted on sixty occasions; in fifty-three of these the tumour was removed and the patient cured, in four improvement resulted but a persistent fistula excluded admission into the former group, and three died. The group "cured" includes those patients in whom a small tag of tumour had to be left attached to an important organ provided the wound was firmly healed, no fistula had resulted, and no symptoms were complained of. Thus, Garre (1918), operating upon Naegeli's patient, left behind a small portion of the cyst wall that was attached to the left auricle and Moir (1936) had to sacrifice a similar portion tightly bound to the pericardium. Likewise, the pedicle that is occasionally encountered running from the sternum or mediastinum to the cyst often has to be ligatured, divided, and the proximal end disregarded. The risk of malignancy supervening in this minute fraction retained by the host can be considered infinitesimal.

The impressive figure of fifty-three patients cured out of sixty operations performed must be viewed in its right perspective and cannot legitimately be placed on a percentage basis. It is natural that the surgeon should regard a successful operation of this sort as a technical triumph worthy of publication and it may well be that many of the failures were never recorded because it was believed they lacked originality and value. Even with this reservation, however, the published experience of thoracic surgeons in all quarters of the globe in this disease has conclusively demonstrated the superior value of complete extirpation over all other procedures and, as this experience widened, engendering a correspondingly wider margin of operative safety, the belief that surgery should be preventive rather than curative in design swiftly gained ground. Thus, in 1924, Aurousseau wrote: "*Le traitement est essentiellement un traitement chirurgical ses indications sont formelles et il n'existe pas de contre indication.*" Harrington's opinion was expressed in 1937 in these unminced words: "The only treatment that is effective for anterior mediastinal tumours is their complete surgical removal and this should be accomplished as soon as the diagnosis can be established, regardless of the severity of the symptoms." With such authoritative statements as these in mind and in the light of the published records, there will be little dispute as to what is the fundamental aim of surgery in this disease. There are, however, certain practical details, due observance of which are important if the patients' chances are to be accurately assessed and success is to be attained.

In the first place it is clear that the position of the tumour, as well as its size, must be taken into account when the prospects of surgery are being reviewed. They can be conveniently divided into the three centage of oxygen in the air breathed, to counteract immediate and delayed shock by blood transfusion, to remove the effusion, sterile or infected, that collects in the pleural or dead space, and, lastly, to promote reinflation of lung and maximum respiratory function. They form the closing chapter and illustrate, with what has gone before, how the scientific and technical advances of recent years have simplified the diagnosis of this disease and placed the successful management of it in the hands of the thoracic team.

SUMMARY

1. A review is presented of dermoid cysts and teratomata of the *mediastinum based upon a study of the world's literature and six* examples that have been recorded for the first time in this communication.

2. After an historical introduction and an account of the six original cases, the clinical features, special investigations used in establishing the diagnosis, the differential diagnosis, complications, and treatment of this class of tumour are considered. The results of treatment are discussed.

3. In an appendix is given a list of references containing case reports from the first recorded example in 1827 until the end of 1939, during which period it has been computed that there are 245 cases on record. This figure does not include those described here.

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Appendix

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CLOSED INTRAPLEURAL PNEUMONOLYSIS

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INTRODUCTORY STATEMENT

THIS study deals primarily with the operative and postoperative complications and mechanical results of pneumonolysis and with early postoperative results of pneumothorax. It is based on my own experience as well as on data obtained from a series collected from the literature, which together with my series totals more than 7,929 operative stages on 7,157 tuberculous patients. In the course of this study the available American and English literature on closed intrapleural pneumonolysis published since Moore's[14] report of a collected series of 2,043 cases in 1934 was reviewed and essential data incorporated into Tables X, XI, and XII.

I agree with those who regard thoracoscopy and pneumonolysis as an integral part of a program of collapse by pneumothorax. It is my opinion that, as will be explained, only the technical results of the operation and complications attributable to it may be regarded as true results of the operation. The so-called clinical results embracing cavity and sputum status are, strictly speaking, the result of postoperative collapse by pneumothorax and not of pneumonolysis. Therefore, it is believed to be not premature to report the technical results, postoperative complications, and clinical course in my series, despite the fact that only six months have elapsed from the date of the last operation.

In Moore's collected series no mention of the preoperative duration of pneumothorax and of the extent of the pulmonary disease was

TABLE I

PREOPERATIVE STATUS

48 PATIENTS

Sex	Males	20
	Females	28
Age	Range	14 to 45
	Average	26
Race	Caucasian	23
	Noncaucasian	25
Pneumothorax	Unilateral	44
	Bilateral	4
Duration of pneumothorax (months)	Range	1 to 38
	Average	8
Extent pulmonary tuberculosis (N. T. A.)	Moderately advanced	11 (23%)
	Far advanced	37 (77%)

From the Olive View Sanatorium, Olive View, Calif.

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TABLE II

HOMOLATERAL LUNG
(50 LUNGS)

Lung	Right		25
	Left		25
Infiltration	Exudative		36 (72%)
	Fibrotic		1
	Mixed		12 (28%)
	None		1
X-ray evidence of cavitation	Demonstrated		39 (78%)
	Not demonstrated		11 (22%)
Size of pulmonary cavities (cm.)	Range		1 to 7
	Average		2¼
	3 cm. or less		29 (74%)
	Over 3 cm.		10 (26%)
Number of cavities per lung	Range		1 to 5
	Average		1½
Known age of cavities (months)	Range		2 to 20 plus
	Average		6½ plus
Pleural effusion (excluding those occupying bottom of costophrenic angle	Serous	Small	3 (12%)
		Moderate	3
	None		44 (88%)
Obliterative pleuritis (positive intrapleural pressures)	Present		3 (6%)
	Absent		47 (94%)

TABLE III

MISCELLANEOUS DATA

Contralateral lung	Entirely clear		6 (13%)	
	Infiltration only (small or moderate)		29 (60%)	
	Patent cavitation		11 (23%)	
	Cavitation controlled by pneumothorax		2 (4%)	
Tubercle bacilli in sputum or gastric washings	Demonstrated	Concentration	28	36 (75%)
		Culture only	8	
	Not demonstrated (but previously demonstrable)	Concentration only	3	12 (25%)
		Culture	6	
	Never demonstrated (Mantoux positive)		3	
Bronchoscopic findings (main and lobar bronchi)	Erythema		4 (14%)	
	Ulceration		0	
	Stenosis		0	
	Negative		24 (86%)	
	Total bronchoscoped		28 (100%)	
Temperature	Febrile (subacute serous effusion)		1	
	Afebrile		49	
Homolateral phrenic paralysis			3	

made.[33] In my collected series the preoperative duration of pneumothorax was found reported for about two-fifths of the patients and it ranged from one to fifteen months; the extent of pulmonary disease was found reported for only about one-fifth of the patients and its incidence was as follows: minimal 6 per cent, moderately advanced 33 per cent, far advanced 61 per cent.

No data regarding the sputum status and the incidence of contralateral or extraparenchymal source of positive sputum were presented with Moore's collected series.[33] In my collected series the sputum status was found reported for about one-half of the patients and was as follows: positive 83.5 per cent, negative 16 per cent, unknown 0.5 per cent. The incidence of contralateral or extraparenchymal source of positive sputum was found reported for about one-eighth of the patients in my collected series and it ranged from 13 to 83 per cent.

THORACOSCOPY AND PNEUMONOLYSIS

This study includes all pneumonolyses, consecutive and unselected, done by me at the Olive View Sanatorium between September, 1941, and October, 1942. They totaled 55 operations on 50 lungs of 48 patients; upon 5 patients the operation was done in two stages. In addition, there were 4 thoracoscopies without pneumonolysis done and these were included in a study already reported.[20]

In 1934, Moore[33] published his study of a series consisting of 2,043 cases collected from the literature of 1924 to 1932, inclusive; the number of operative stages was not stated. My collected series consists of 5,114 patients subjected to more than 5,886 operative stages done between the years of 1927 and 1942, and reported in the literature of 1933 to 1943, inclusive; in this series the year of operation was indicated for about two-thirds and the number of operative stages for about four-fifths of the total number of patients.

As a rule I found more adhesions thoracoscopically than expected from the study of the roentgenograms, including stereoscopic films, supporting earlier contentions that thoracoscopy and not roentgenography is the only accurate method of determining the exact number, character, and feasibility of freeing adhesions.[20] In the great majority of cases the visceral attachment of adhesions freed was on the upper lobe and the parietal attachment on the upper posterior chest wall.

In Moore's collected series galvanocautery was employed by 83 per cent, electrosurgery by 7 per cent, and the combination of the two methods by 10 per cent of those reporting.[33] The type of current used in my collected series was indicated for about three-fourths of the patients: galvanocautery 41 per cent, electrosurgery 26 per cent, combination of the two methods 3 per cent, and a succession of different methods 30 per cent. The following synonymous terms were encountered in the literature reviewed in connection with the description of type of current employed in pneumonolysis: for galvanocautery: cautery, actual cautery, electrocautery; for electrosurgery: high frequency current, endothermy, surgical diathermy. The two-cannula technique and galvanocautery were employed in my series.

The patients were kept in the surgical wards for a period varying from one to several weeks after I had operated upon them. They were fluoroscoped on the first postoperative day and as indicated thereafter. About one-half of the patients were under my own care while in the surgical wards; upon return to the medical wards these patients came under the care of the medical ward physicians.

TABLE IV

Duration of operation (min.)	Range	20 to 110 min.
	Average	60 min.
	30 min. or less	10 operations (18%)
	60 min. or less	36 operations (65%)
	Over 60 min.	19 operations (35%)

No mention of the duration of the operations was made in Moore's collected series,[33] in my collected series this was found reported for about one-fifth of the operations and it ranged from a few to ninety minutes with a maximum average of sixty-five minutes.

TABLE V

Type of adhesion operated upon	Strings	17 (10%)
	Cords	57 (34%)
	Bands	92 (56%)
	Total	166 (100%)
Number of adhesions operated on (per patient)	Range	1 to 9
	Average	3

In anticipation of a correlation between the type of operation and complications and results, I classified all operations as follows: complete pneumonolysis is one involving complete freeing of all adhesions attached to the diseased pulmonary lobe; incomplete is one involving

TABLE VI

Type of operation	Complete	30 (55%)
	Incomplete	8 (15%)
	Partial	17 (30%)

TABLE VII

POSTOPERATIVE INSTITUTIONAL FOLLOW-UP (AS OF APRIL 1, 1943)

Discharged from Olive View Sanatorium	15 (31%)
Still at Olive View Sanatorium	33 (69%)
Died at Olive View Sanatorium	0

complete freeing of only some of the adhesions attached to the diseased lobe; and finally, partial pneumonolysis is one involving partial freeing of all or some of the adhesions to the diseased lobe.

The postoperative stay at the Sanatorium ranged from one week to eighteen months and averaged eight and one-half months. This is shown in greater detail in Table VIII.

TABLE VIII

LENGTH OF STAY AT SANATORIUM	NO. OF LUNGS	PER CENT
Less than 1 mo.	1	2
Less than 3 mo.	5	10
Less than 6 mo.	11	22
Less than 10 mo.	29	58
More than 10 mo.	21	42
Total	50	100

In 5 of the patients still at the Sanatorium, pneumothorax had been supplemented by phrenic nerve interruption, and in 9 patients it was abandoned as ineffective three weeks to eleven months postoperatively (average of four months) and a thoracoplasty substituted or planned on. Disease in the contralateral lung was treated as indicated. At the time of this communication, only one patient is clinically worse than she was at the time of operation which was complete and free of complications, but due to ulcerative and stenotic tuberculous bronchitis the pulmonary cavity is still patent, the sputum positive, and she is febrile. Since the operation 6 patients were bronchoscoped for the first time and 5 others were rebronchoscoped; 2 of the former and 1 of the latter were found to have bronchial ulceration.

In Moore's collected series the duration of the postoperative period of observation was not specified except for the statement that only immediate results were given and that Unverricht was the only one to report results following re-expansion.[31] In my collected series the length of the postoperative period of observation was found reported only for about one-half of the patients and it ranged from one to sixty months; Jones was the only one to report results following re-expansion in part of his series.[19]

POSTOPERATIVE COMPLICATIONS

For the sake of completeness all complications, including fever and small effusions, which occurred during the entire postoperative stay at the Sanatorium regardless of whether attributable to the operation or to coexisting tuberculosis and pneumothorax were included (Table IX). Only complications which were not present preoperatively were included in my own series; wherever sufficient data were available the same was done in my collected series.

type of current employed in pneumonolysis: for galvanocautery: cautery, actual cautery, electrocautery; for electrosurgery: high frequency current, endothermy, surgical diathermy. The two-cannula technique and galvanocautery were employed in my series.

The patients were kept in the surgical wards for a period varying from one to several weeks after I had operated upon them. They were fluoroscoped on the first postoperative day and as indicated thereafter. About one-half of the patients were under my own care while in the surgical wards; upon return to the medical wards these patients came under the care of the medical ward physicians.

TABLE IV

Duration of operation (min.)	Range	20 to 110 min.
	Average	60 min.
	30 min. or less	10 operations (18%)
	60 min. or less	36 operations (65%)
	Over 60 min.	19 operations (35%)

No mention of the duration of the operations was made in Moore's collected series,[33] in my collected series this was found reported for about one-fifth of the operations and it ranged from a few to ninety minutes with a maximum average of sixty-five minutes.

TABLE V

Type of adhesion operated upon	Strings	17 (10%)
	Cords	57 (34%)
	Bands	92 (56%)
	Total	166 (100%)
Number of adhesions operated on (per patient)	Range	1 to 9
	Average	3

In anticipation of a correlation between the type of operation and complications and results, I classified all operations as follows: complete pneumonolysis is one involving complete freeing of all adhesions attached to the diseased pulmonary lobe; incomplete is one involving

TABLE VI

Type of operation	Complete	30 (55%)
	Incomplete	8 (15%)
	Partial	17 (30%)

TABLE VII

POSTOPERATIVE INSTITUTIONAL FOLLOW-UP
(AS OF APRIL 1, 1943)

Discharged from Olive View Sanatorium	15 (31%)
Still at Olive View Sanatorium	33 (69%)
Died at Olive View Sanatorium	0

TABLE X

INCIDENCE OF COMPLICATIONS ATTRIBUTABLE TO PNEUMONOLYSIS

More Than 7,929 Operative Stages on 7,157 Patients From 1924 to 1943 Inclusive

COMPLICATIONS	INCIDENCE (IN TERMS OF PER CENT)					DATA AS TO INCIDENCE IN GOORWITCH COLLECTED SERIES WERE FOUND REPORTED ONLY FOR ABOUT
	IN SERIES COLLECTED BY				GOORWITCH—OWN SERIES, 1941-1942 (48 PATIENTS)	
	MOORE, 1924-1932 (2,043 PATIENTS)		GOORWITCH, 1933-1943 (5,114 PATIENTS)[1]			
	RANGE	AVERAGE	RANGE	AVERAGE		
Entirely absent	?	?	27-89	49	89	1/10 of operations
Present	?	?	11-73	51	11	
Prolonged fever	?	?	0-13	7	8.6	1/4 of operations
Intrapleural hemorrhage						
Small	0-100[2]	0.85[2]	0-12	3.30	0	1/6 of operations
Large or serious	0-100[2]	0.80[2]	0-13	1.70	1.8	2/3 of operations
Severe surgical emphysema	?	?	0-20	2.00	1.8	2/5 of operations
Spontaneous pneumothorax	?	?	0-12	2.30	0	2/5 of operations
Bronchopleural fistula	?	?	0- 8	2.50	0	1/2 of operations
Serous pleural effusion						
Small	0- 93[3]	25.50[3]	0-55	10	5.4	1/4 of operations
Large	0- 33[4]	8.80[4]	0-27	9.50	3.6	1/3 of operations
Empyema						
Pure tuberculous	0- 8	2.20	0-24[5]	3	0	2/3 of operations
Pyogenic	?	?	0- 6	0.24	0	1/2 of operations
Mixed tuberculous	0- 23	1.80	0- 5	1	0	1/2 of operations
Obliterative pleuritis	?	?	0-11	7	0	1/4 of patients
Loss of pneumothorax	0- 4	0.29	0-10	3	0	1/5 of patients
Air embolism	0- 0.04	0.04	0- 1.1	0.06	0	1/6 of operations
Pleurocutaneous fistula	0- 8	0.14	0- 2	0.35	0	1/7 of operations
Secondary perforation	0- 4	0.39	0- 1.3	0.70	0	1/12 of operations
Extension of pulmonary tuberculosis	?	?	0- 6	1.30	0	1/6 of operations
Nerve injury	?	0.04	0	0	0	Very rarely
Shock	?	0.04	0- 2	0.15	0	
Death	0-100[6]	1.08[6]	0- 6	1.18	0	9/10 of patients

[1] Including Goorwitch's own series.

[2] Reported only for 1,883 cases.

[3] Reported only for 1,807 cases.

[4] Reported only for 1,875 cases.

[5] Turner reported an incidence of 24 per cent by including all those cloudy and purulent any time postoperatively;[28] 0 to 10 per cent is the more correct range.

[6] Reported only for 1,850 cases.

Fever.—Unless high and prolonged or accompanied by other significant complications, fever is not a complication in the strict sense of the word, but is considered here, as in previous studies,[20, 21] only for the sake of completeness. Temperature of 100° F. and over was again arbitrarily chosen to designate fever of some clinical significance in intrapleural surgical intervention.

Accordingly, in my own series fever was present following 24, or 44 per cent, operations; its onset was on the day of operation or on the first postoperative day and it lasted continuously from one day to three weeks and an average of four days. In 75 per cent of these patients the duration of fever was three days or less, in 92 per cent one week or less, and in the remaining 8 per cent over 1 week (maximum of three weeks). Maximum* fever ranged from 100 to 102° F. and averaged 100.4° F. Following 15 operations, maximum fever was 100, following 7 operations 101, and following 2 operations 102° F. Thus, fever was not a serious complication, as among those febrile it did not exceed 100° F. in 36 per cent, was the only complication in 75 per cent, and lasted one week or less in 92 per cent.

In his collected series, Moore[33] did not mention fever among the postoperative complications. In my collected series the incidence of prolonged fever in one-fourth of the operations for which it was reported was 7 per cent. In my own series the incidence of prolonged fever (exceeding 100° F. and lasting over one week) was 3.6 per cent.

Intrapleural Hemorrhage.—I assume that loss of up to two ounces of blood, either from trocar wounds or cauterized adhesions, should not be regarded as a hemorrhage.[21] No criteria of what constitutes a small hemorrhage were found in the literature reviewed. If transient, small intrapleural hemorrhage is of no clinical significance. Large intrapleural hemorrhage is a fairly rare but, depending on its source, a most dangerous complication. In my single case of this complication the approach to the adhesions in the extreme apex was exceptionally difficult and more than two trocar punctures were made; slight oozing from one of the trocar wounds was noted during the operation but was thought to be of no importance. No bleeding from the released adhesions was noted and the patient left the operating room in excellent condition; however, during the first postoperative week a total of about 500 c.c. of at first sanguineous and later serosanguineous fluid was aspirated. The source of hemorrhage must have been an intercostal vein which was traumatized by the trocar and which continued to ooze after the withdrawal of the cannula.

In Moore's collected series one of the 31 hemorrhages proved to be fatal; it was described as small but was responsible for one of the 19 deaths attributable to the operation.[33] In my collected series 8 of the 159 hemorrhages proved to be fatal, thus accounting for 8 of the 59 deaths attributable to the operation; they were described as large (Table XI).

Although intercostal vessels constitute the most frequent source of intrapleural hemorrhage and even death is known to have resulted from uncontrollable hemorrhage from an intercostal artery, massive hemorrhage from injury to the great mediastinal vessels or the lung itself is by far the more serious. I have personal knowledge of three instances of operative injury to the mediastinal vessels with a rapidly fatal outcome despite a thoracotomy in one of them. Only infrequently was the source of hemorrhage stated in the literature reviewed.

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*Fractions of a degree were eliminated by substituting the nearest whole number; thus 100.4 was recorded as 100° F. and 100.6 as 101° F.

TABLE XI

CAUSES OF DEATH ATTRIBUTABLE TO PNEUMONOLYSIS

CAUSES OF DEATH	SERIES COLLECTED BY	
	MOORE* (1924-1932)	*GOORWITCH*† INCLUDING HIS OWN SERIES (1933-1943)
Spontaneous pneumothorax	?	5
Bronchopleural fistula	?	24
Empyemas		
Pure tuberculous	?	1
Pyogenic	?	2
Mixed tuberculous	"majority"	5
Intrapleural hemorrhage	1	8
Air embolism	1	1
Extension of pulmonary disease	?	2
Cardiorespiratory decompensation	?	1
Unspecified	?	4
Total mortality	19 (1.08%)	53 (1.18%)

*1,850 cases.

†5,114 cases, but fatality statistics found reported only for about nine-tenths of the series.

Moore[33] stated that nearly all of the reported hemorrhages occurred with the use of the cautery, but when it is realized that 83 per cent of those reporting employed the cautery it will appear that the type of current used was not the deciding factor.

Surgical Emphysema.—Mild subcutaneous emphysema localized to the chest wall about the trocar wounds is common; it is not clinically important and strictly speaking is not a complication. In my own series there was one instance of severe subcutaneous emphysema in a patient with extensive pulmonary disease and a severe cough which was not controlled properly; loss of pneumothorax did not result.

Spontaneous Pneumothorax.—Spontaneous pneumothorax should be regarded as a serious complication as it may be followed by a bronchopleural fistula and a mixed tuberculous empyema. In my collected series 9 per cent of all deaths attributable to the operation resulted from this complication (Table XI). In my own series spontaneous pneumothorax not attributable to the operation occurred twice in the same patient three and five months postoperatively; it was relieved by simple aspiration of air but was finally abandoned when seven months postoperatively the 7 cm. cavity was still patent in spite of complete and uneventful pneumonolysis.

Bronchopleural Fistula.—The danger of the complication of bronchopleural fistula when clinically demonstrable is exceeded only by massive intrapleural hemorrhage from the great mediastinal vessels. No mention of this complication was made by Moore,[33] who probably included it in the mixed tuberculous empyema group of his collected series.

When clinically demonstrable a bronchopleural fistula is almost always accompanied or followed by mixed tuberculous empyema suggesting that the source of the secondary infection is in the traumatized or sloughing lung. The incidence of this dreadful complication could undoubtedly be reduced if Newton's[34] ably presented concept of releasing as opposed to cutting, dividing, or severing adhesions were more fully appreciated and if adhesions only questionably operable as determined thoracoscopically were left alone. The opening statement in the preceding paragraph is amply supported by the fact that in my collected series about two-fifths of these complications proved fatal and a little less than one-half of all deaths attributable to the operation resulted from this complication (Table XI).

Pleural Effusions.—Most unoperated pneumothoraces have fluid at one time or another. Head is quoted by Moore[33] as having stated that following pneumonolysis tuberculous effusions were twice as common as in unoperated pneumothoraces; this must have been true two decades ago. In Moore's collected series the incidence of small and large pleural effusions was about 34 per cent,[33] in my collected series 25.5 per cent, and in my own series only 9 per cent.

Prominent in the more recent literature was evidence that pneumonolysis was not an important factor in the production of pleural effusions and that it certainly did not add materially to the incidence of pleural effusions occurring commonly with pneumothorax alone. In fact, purulent fluids and large serous effusions are believed by these workers to be less frequent following pneumonolysis than in unoperated pneumothoraces restricted by adhesions.[11, 12, 21, 29]

Pure Tuberculous Empyema.—Both purulent and large serous effusions complicating induced pneumothorax and closed intrapleural pneumonolysis are believed to originate in spontaneous or operative ulceration of pleural or subpleural tuberculous foci.[19] As already stated, the more recent reports indicate that following closed intrapleural pneumonolysis the incidence of empyema is no greater than in unoperated pneumothoraces.[11, 12, 21, 29]

In my collected series death from empyema occurred in only 1 of the 114 patients with this complication; in the 1 patient in my own series who was febrile at the time of the operation because of a subacute serous effusion a small amount of clear fluid persisted postoperatively and after many months of neglect to aspirate was found to have become purulent.

Mixed Tuberculous Empyema.—As previously stated, mixed tuberculous empyema is a very serious and fairly constant sequel to bronchopleural fistula and the two follow spontaneous or operative trauma to tuberculous lung tissue. In Moore's collected series all mixed tuberculous empyemas resulted from perforation of lung or opening into a pulmonary cavity and about one-third of them died; this complication was responsible for the majority of all deaths in his series.[33] In my collected series bronchopleural fistula or tuberculous, pyogenic or mixed tuberculous empyema, all of which represent pulmonary trauma associated with pleural infection, proved fatal in one-sixth of the cases and were responsible for a majority of all deaths. (Table XI).

Pyogenic Empyema.—It is generally thought that external contamination, as by unsterile instruments, is much less frequently responsible for this complication than operative trauma to lung tissue. Its outcome was fatal in 2 of the 7 instances in my collected series (Table XI).

Obliterative Pleuritis.—By the term obliterative pleuritis is meant a gradual restriction of the pleural space through creeping out of the lung edges accompanied by a rise of intrapleural pressures and decreased demand for air refills. Following pneumonolysis the incidence of this complication is said to be definitely higher than in unoperated pneumothoraces.[29] The only instance of obliterative pleuritis in my series developed eight months postoperatively without loss of space and was not attributed to the operation.

Other Less Common Complications.—Less common complications were found reported but rarely in my collected series and were not encountered in my own series. They include loss of pneumothorax space, air embolism, pleurocutaneous fistula, secondary perforation of the lung, shock, nerve injury, and extension of pulmonary disease.

As an early postoperative complication, loss of pneumothorax space is generally employed to designate loss of pneumothorax from rapid loss of air through severe and uncontrollable surgical emphysema and not through gradual obliterative pleuritis. Air embolism was uniformly fatal. In Moore's collected series an effusion was present at the time of operation in those cases which became complicated by pleurocutaneous fistula. He regarded perforation of lung secondary only if it occurred more than six weeks postoperatively.[33]

Operative and Postoperative Deaths.—In Moore's collected series the majority of the 19 deaths attributable to the operation was ascribed to mixed tuberculous empyema resulting from perforation of the lung or opening into pulmonary cavities within the first six postoperative weeks. Only 1 fatality was ascribed to air embolism and 1 to a small intrapleural hemorrhage despite the fact that there were 15 serious hemorrhages; the causes of the remaining deaths were not given.[33]

In my collected series also the majority of the 53 deaths attributable to the operation resulted from lung trauma and infection; second in frequency was intrapleural hemorrhage and the remaining deaths were due to less common and unspecified causes. Thus, operative trauma to lung with resulting infection of the pleural cavity is by far the most common cause of death attributable to the operation and is probably due more frequently to defective judgment and failure to recognize inoperable adhesions than to a pure accident. On the other hand, intrapleural hemorrhage, the second most common cause of

AUTHOR AND DATE OF PUBLICATION	OPERATIVE TECHNIQUE	NUMBER OF PATIENTS	PREOPERATIVE STATUS				OPERATIVE AND POSTOPERATIVE COMPLICATIONS (%								
			N. T. A. CLASSIFICATION (%)		SPUTUM (%)		INTRAPLEURAL HEMORRHAGE		SPONTANEOUS PNEUMOTHORAX	BRONCHOPLEURAL FISTULA	SEROUS EFFUSIONS		EMPYEMA		
			M.A.	F.A.	+	−	SMALL	LARGE OR SERIOUS			SMALL	LARGE	PURE TUBERCULOUS	PYOGENIC	MIXED TUBERCULOUS
Phillips & Knoepp	?	26	?	?	?	?	?	?	4	?	?	?	?	?	?
Scarborough	G	77	?	?	?	?	3	0	?	0	7		0	1	0
Turner (1939)	E	59	?	?	?	?	?	0	?	8	?	8	24	?	?
Grimes	?	19	?	?	?	?	Few	5	?	?	10		10	?	?
Newton	E	146	?	44	75	25	0	0	?	0	12	5	2	0	0
Stemmerman & Tchertkoff	?	126	?	Majority	100	0	0	0	2.4	5	?	?	2.4	0	5
Stocklen & Greene	?	833	?	?	?	?	?	?	?	?	?	?	?	?	?
Wollaston (1940)	G	200	?	?	78	22	11	1.5	?	0.5	?	13	2	0	0
Black	?	69	13	84	80	20	?	1.1	?	?	Few		1.1	?	?
Hoffman & Brantigan	G	109	?	?	?	?	1	0	2	?	?	?	5	1	2
McCullough & Chapman	G	111	?	?	100	?	0	1.8	0.8	0		?	6	?	0
Thompson & Greenberg (1941)	?	39 92	?	?	100 70	0 24	?	2.6 3.3	?	?	8 11		?	?	0
Carp & Kornblith	G 5% E 95%	75	?	?	84	16	?	2.7	1.3	?	30		2.7	0	0
Hedberg	G	68	37	59	58	42	6	0	?	?	17	5	2.4	0	0
Jones, J. C. (1942)	G	36	?	?	100	0	0	0	0	0	55	0	0	0	0
Olive View Sanatorium series collected by Goorwitch	G 55% E 45%	365	?	?	66	31	3	6	4	3	7	7	2	0	2
Dickey	–	139	–	–	–	–	–	–	–	–	–	–	3.6	–	–
Goorwitch (1943)	G	48	23	77	75	25	1.8	1.8	0	0	5.5	3.6	0	0	0
Total (Goorwitch's collected series), 1944 (1933-1943)	G 41% E 26% C 3% M 30%	5,114	33	61	83.5	16	3.3	1.7	2.3	2.5	16	9.5	3	0.24	1

XII—CONT'D

		OPERATIVE RESULTS (%)										MISCELLANEOUS
		CAVITY		SPUTUM		PNEUMONOLYSIS						
								TECHNICALLY		CLINICALLY		
LOSS OF PNEUMOTHORAX SPACE	DEATH	CLOSED	PATENT	CONVERTED TO NEGATIVE	UNCONVERTED	COMPLETE	INCOMPLETE	SUCCESSFUL	UNSUCCESSFUL	SUCCESSFUL	UNSUCCESSFUL	
?	4	?	?	?	?	?	?	?	?	*80*	*20*	
1.3	0	72	28	?	?	?	?	74	26	79	21	Average preoperative age pneumothorax 6 mo.; complete absence of complications 80%; no pleurocutaneous fistula or extension of T.b.
?	3.3	87	13	?	?	?	?	88	12	76	24	Average preoperative age pneumothorax 4 mo.; contralateral source T.b. 32%; operative time few to 90 min.
?	5	?	?	?	?	?	?	84	16	79	21	No pleurocutaneous fistula
0	0	?	?	85	15	81	19	-	-	88	12	P.O. period of observation for complications only; no embolism or shock or pleurocutaneous fistula
?	0	53	47	54	46	?	?	?	?	54	46	Contralateral source of T.b. 47%; no shock
?	0.5	75	25	?	?	?	?	?	?	?	?	
?	0.5	90	10	87	13	?	?	96	4	96	4	Preoperative age pneumothorax 1½-2 mo.; average operative time 60 min.; secondary perforation 0.5%; complete absence of complications 71%
?	1.4	?	?	75	25	?	?	81	19	?	?	
?	2	?	?	?	?	?	?	?	?	?	?	Average operative time 60 min.
0	0.8	69	31	43	57	?	?	?	?	?	?	Preoperative age of pneumothorax 1-8 mo.; contralateral source T.b. 18%; no extension of T.b. or pleurocutaneous fistula
?	$\frac{0}{1.1}$	?	?	$\frac{90}{71}$	$\frac{10}{29}$	?	?	?	?	?	?	Average preoperative age pneumothorax $\frac{1\frac{1}{2}}{6}$ mo.; complete absence complications $\frac{82\%}{75\%}$
?	4	44	56	41	59	43	57	-	-	40	60	Contralateral source T.b. 83%; complete absence complications 27%; extension of T.b. 4%; no pleurocutaneous fistula
?	0	86	14	87	13	?	?	?	?	93	7	Preoperative age of pneumothorax 1½-2 mo.; *contralateral source T.b. 13%*
0	0	?	?	94	6	50	50	-	-	94	6	Contralateral source T.b. 35%; no nerve injury or pleurocutaneous fistula
?	3	?	?	?	?	30	70	-	-	?	?	Average preoperative age pneumothorax 9 mo.; average operative time 65 min.; complete absence complications 84%; extension T.b. 2%; no air embolism
-	-	-	-	-	-	-	-	-	-	-	-	From a paper on T.b. Empyema
0	0	76	24	76	24	55	45	-	-	?	?	Average preoperative age pneumothorax 8 mo.; contralateral source T.b. 23+%; average operative time 60 min.; complete absence complications 56%; no extension T.b., shock, nerve injury, air embolism or secondary perforation
3	1.18	72	28	73	27	52	48	90	10	73	27	Operative stages 5,886; date of operations 1927-1942; duration of P.O. observation 1-60 mo.; minimal T.b. 0%; prolonged fever 7%; severe subcutaneous emphysema 2.6%; obliterative pleuritis 7%

title given this section of the manuscript. The postoperative clinical results of pneumothorax in my own series will be presented under the headings of Cavity Status and Sputum Status.

In the absence of restricting adhesions we regard a clinically unsuccessful *pneumothorax as a failure due to factors other than mechanically* ineffective collapse. Why blame the operation when, in the absence of postoperative complications, pneumothorax remains a clinical failure despite conversion by means of complete pneumonolysis from mechanically defective into mechanically perfect collapse? It is of the pneumothorax that we expect cavity closure and sputum conversion. All pneumonolysis should be expected to achieve is concentrically free collapse which is our aim when pneumothorax is first induced. That if pneumothorax is indicated at all it should be free of restricting adhesions is concluded on the basis of past experience of those who observed that superior results, both immediate and ultimate, were obtained in patients with concentrically free collapse either spontaneous or brought about by pneumonolysis.[24, 31, 33, 43, 44]

I believe it unfair to the procedure of pneumonolysis to judge it by the subsequent clinical results of pneumothorax or to compare it to phrenic nerve interruption. It is true that in the presence of pneumothorax neither a technically successful phrenic nerve interruption resulting in paralysis and elevation of the hemidiaphragm nor a complete pneumonolysis resulting in concentrically free collapse is synonymous with clinical success of the pneumothorax, but this is as far as the similarity of the two operative procedures goes. Phrenic nerve interruption is not a substitute for pneumonolysis. Close analysis reveals that phrenic nerve interruption does not alter the character of pneumothorax restricted by adhesions and is an independent procedure apart from pneumothorax and therefore entitled to its share of credit or blame for clinical results. Complete pneumonolysis, on the other hand, radically alters the character of the collapse by pneumothorax and as an integral part of collapse by pneumothorax is not entitled to its own clinical results; the clinical results of collapse are those of pneumothorax and not of pneumonolysis. The results of pneumonolysis consist of mechanical alterations in collapse and of operative and postoperative complications.

Cavity Status.—In my collected series cavity status following pneumonolysis was found reported for about one-half of the patients and cavity closure ranged from 44 to 90 per cent and averaged 72 per cent. In my own series of 50 lungs there were 11 lungs with no preoperative x-ray evidence of cavitation, 1 lung with insufficient x-ray follow-up, and 4 with definitely insufficient operative freeing of the lung (done early in my experience) to expect any alteration in the character of the collapse. In the remaining 34 lungs cavity closure was obtained in 74 per cent and some or all of the cavities persisted in 26 per cent.

Insofar as pulmonary cavitation is concerned, the aim of the operation is to alter the collapse to bring about cavity closure as speedily as possible. In my series the first roentgenogram was made one week postoperatively in the great majority of cases; in a few it was made within the first three postoperative days, and in several one to three months postoperatively. Subsequently patients were x-rayed at two- to three-month intervals. It was therefore impossible to establish accurately the actual interval between operation and cavity closure. However, with the data in hand an attempt was made to determine the cavity status in the first postoperative roentgenogram as well as in the latest, and to establish the date of cavity closure. Thus, in the first postoperative roentgenogram of the 34 lungs all cavities were closed in 18, or 53 per cent, some of the cavities closed in 3, and all cavities persisted, although smaller, in 13. In the final roentgenograms the findings were: 25, or 74 per cent, 2 and 7, respectively. Earliest x-ray evidence of cavity closure was found in four days to seven months and an average of 1.7 months postoperatively; no relationship was noted between the type of operation and the time of closure. Carp and Kornblith[5] reported cavity closure in an average of about one month following complete, and two months following partial, pneumonolysis. Some of the lungs in my series were subsequently re-expanded and again collapsed by thoracoplasty and in these cavity status was given as of the date of discontinuance of pneumothorax.

Sputum Status.—In my collected series sputum status following operation was found reported for about one-half of the patients and sputum conversion ranged from 41 to 95 per cent and averaged 73 per cent. In my own series of 48 patients there were 12 whose sputum was negative preoperatively, 12 with demonstrable sources of positive sputum other than cavitation in the homolateral lung (contralateral cavitation, laryngeal and/or tracheobronchial ulceration), and 3 with definitely insufficient operative freeing of the lung (done early in my experience) to expect any alteration in the character of the collapse. In the remaining 21 patients, 7 of whom had no x-ray evidence of cavitation, the sputum was positive in 14 on concentration and in 7 on culture only. Sputum conversion was achieved in 76 per cent of these and it failed in 24 per cent.

One of the chief aims of early pneumonolysis is to alter the character of the collapse to produce sputum conversion as speedily as possible after induction of pneumothorax. In my own small series sputum studies were not made at regular postoperative intervals; it is therefore impossible to establish accurately the earliest date of sputum conversion. The date of the first negative sputum followed by consistently negative sputa was regarded as the date of conversion. During the first postoperative month no sputum examination was done in 9 patients, the sputum remained positive in 5, and it was negative in 7 patients. At the time of discharge or in April, 1943, the sputum was converted in 16, or 76 per cent, patients and unconverted in the remaining 5, or 24 per cent. Of the 5 remaining positive, 3 were positive on concentration and 2 on culture only. Of the 16 in which it was converted, 14 were on the basis of an average of 2 cultures of sputum and/or gastric washings and the remaining 2 on the basis of an average of 4 examinations by the concentration method only.

In my series conversion took place one to seven months and an average of three months postoperatively; in 10, or 63 per cent, it was converted in three months or less and in the remaining 6 in over three months. Without stating their criteria of sputum conversion, Stemmerman and Tchertkoff[44] reported 88 per cent sputum conversion within the first three postoperative months and 12 per cent after this time; Carp and Kornblith[5] reported sputum conversion usually one month following cavity closure and an average of two months postoperatively in complete and three months in partial operation. Employing rigid criteria Jones[29] reported an average of five and one-half months for sputum conversion.

DISCUSSION

Like any other therapeutic procedure, closed intrapleural pneumonolysis should be evaluated by the positive benefit to the patient and by the absence of complications aggravating the patient's condition which the operation was intended to improve. It is realized that in especially desperate circumstances pneumonolysis may be the only procedure offering any hope at all.

Examination of data in Table X will disclose that while some of the series reviewed were remarkably free of serious complications, the incidence for the entire collected series based on complications reported definitely as present or absent was not very low. If failure to mention a particular complication were regarded as its absence, the incidence of complications for the entire collected series would have been lower than that given in Table X. When it is remembered that pneumonolysis is an elective procedure and only rarely one of emergency, it will be realized that there is definite room for reduction in the incidence of the more serious complications. As already pointed out by Newton,[34] the incidence of these complications can be reduced to the minimum by improvement in both operative technique itself and surgical judgment as to operability of thoracoscopically inspected adhesions. Freeing or releasing of adhesions off their parietal attachments as contrasted with cutting, dividing, and severing them would go a long way in preventing serious lung injury.

While none of the postoperative complications in my own series, except for the single instance of intrapleural hemorrhage, were of any consequence, it is of interest to know that among those febrile the incidence of exudative infiltration and pleural effusion at the time of operation and of early, partial or prolonged operation was slightly higher than among those afebrile postoperatively. Among patients with postoperative effusion the incidence of early and partial operation was slightly greater than among those without postoperative effusion. However, the benefit derived from earlier cavity closure and sputum conversion undoubtedly more than outweighed the disadvantage of the minor complications mentioned.

In examining the procedure of pneumonolysis for any positive benefit to the patient, improvement in the mechanical character of the collapse, the only direct result of the operation, is also the only true criterion of benefit. This criterion is supported by the universally recognized observation that collapse free of restricting adhesions is in the great majority of instances synonymous with clinically successful collapse. However, it should also be mentioned that partial or incomplete pneumonolysis is often sufficient to alter the collapse so as to result in clinical success. In my collected series the incidence of clinical success is almost uniformly greater than the incidence of complete pneumonolysis.

The relationship between the type of pneumonolysis and the clinical results of pneumothorax ranged from the statement of Andrews,[3] that clinical success was only slightly more frequent following complete than incomplete operation, to the report of Edwards and Lynn,[16] who obtained successful "radiological results" following complete pneumonolysis two and one-half times as frequently as following partial operation. In attempting to find out why in 7 patients of my own series cavitation persisted despite greater incidence of complete pneumonolysis and smaller incidence of large adhesions, it was discovered that the average known age of cavitation and of pneumothorax was one and one-half times as great and incidence of obliterative pleuritis three times as great as among the 25 in whom cavity closure was achieved. Tuberculous bronchitis did not appear to be a factor in this small series.

A similar attempt to explain failure to convert sputum despite complete pneumonolysis in 5 patients free of contralateral source of positive sputum led to the discovery that the incidence of cavitation and the average known age of cavitation and of pneumothorax were twice as great as among the 16 patients in whom sputum conversion was achieved despite a lower incidence of complete pneumonolysis. Suspicious bronchoscopic findings either before or after operation were much more common among those in whom sputum was not converted than among those who succeeded. These findings indicate that better results from postpneumonolysis pneumothorax may be expected by earlier induction of pneumothorax and earlier pneumonolysis, and in patients free of such complicating factors as obliterative pleuritis and tuberculous bronchitis.

To be truly the optimum time for pneumonolysis it must be established that the technical and clinical results will be better and be obtained sooner, and the complications fewer and less serious if the operation is done at this time than if it is postponed. While there is universal agreement not to go on with a clinically unsuccessful pneumothorax indefinitely, the recommendations regarding the optimum time for pneumonolysis vary considerably. The time recommended in the literature reviewed as the optimum time for pneumonolysis ranged from three weeks to six months following induction of pneumothorax,[5, 12, 24, 29, 33, 48] the trend being definitely in the direction of earlier operation. Others reported that no relationship could be found among preoperative duration of pneumothorax, clinical results, and incidence of postoperative complications[16, 21, 44] and recommended handling each case on its own merits, that is, individualizing.[44]

In his collected series, Moore[33] did not state the preoperative duration of pneumothorax; however, he did recommend three to six months of collapse prior to consideration of pneumonolysis for two reasons: to allow for adhesion stretching and possible cavity closure and to render the pleura less irritable and less likely to react to operative trauma. In discussing Moore's paper in 1933, O'Brien recommended giving pneumothorax at least six months before pneumonolysis is attempted because before that time one cannot tell whether or not cavity will close without pneumonolysis.[33] In discussing Drash's paper in 1938 both Jacobeus and Welles argued that operating within two months of the induction of pneumothorax did not lead to inferior results or increased incidence of complications and that adhesions grew more fibrous, thicker, and shorter with time.[12]

In my own small series here reported serious complications were not encountered, but it was found that failure to obtain cavity closure and/or sputum conversion was definitely associated with pneumothorax of duration one and one-half to two times longer than that associated with clinical success. I agree with those who advocate early thoracoscopy without waiting six months to see whether the adhesions will stretch and the cavity close, provided the pneumothorax space is large enough to make operation feasible.

According to Moore[33] the indication for pneumonolysis is incomplete pneumothorax of three to six months' duration which on account of string, cord, or band adhesions has not produced a satisfactory collapse of diseased lung, that is, cavity closure, sputum conversion, and freedom from distressing cough and pain after refills. The discussants of his paper declared that the presence of patent cavities and adhesions in the roentgenograms did not necessarily mean that the adhesions were holding the cavities open; phrenic paralysis and increased intrapleural pressures were advocated before attempting pneumonolysis because stretching adhesions was not very dangerous and one could not tell during the first six months whether or not cavities would close without pneumonolysis.[33] We still do not know which cavities are held open by the adhesions. But we do know that following release of adhesions the great majority of cavities do close, which suggests that they were prevented from closing by restricting adhesions. And because we do not know which cavities are and which are not held open by the adhesions and because we do know that superior results and fewer complications are obtained in the absence of adhesions, it is believed that the patient deserves a thoracoscopic examination and if the adhesions are found feasible pneumonolysis should be performed. Apparently it was this concept evolved during the past decade that was responsible for the gradual but steady liberalization of the indications for pneumonolysis, first through earlier operative intervention in clinically unsatisfactory pneumothorax[5, 12, 16, 21, 24, 29, 44, 45] and later through intervention even in symptom-free patients provided the operation can be done safely and without obvious risks.[12, 21, 23, 24, 29, 31, 34, 43, 44, 45]

SUMMARY AND CONCLUSIONS

The available English and American literature on closed intrapleural pneumonolysis published since the publication of Moore's comprehensive report in 1934 was analyzed and important data tabulated (Tables X, XI, XII). My own experience with a series of 55 operative stages on 48 consecutive and unselected patients was presented (Table IX). This small series together with those found in the literature reviewed, excluding Moore's collected series of 2,043 cases, totaled more than 5,886 operative stages on 5,114 patients.

Operative technique, complications attributable to the operation, technical results of the operation, and clinical results of pneumothorax following operation constitute the chief subject of this study. The consensus appears to be that the two-cannula technique is superior to the single cannula technique and although electrosurgery has gained considerably in popularity during the past decade, galvanocautery and the combination of the two has as many or more adherents than electrosurgery.

Criteria for attributability of complications to the operation were formulated and all such complications analyzed. It was from a preliminary study of my own small series that the impression was gained that the very great majority of postoperative complications attributable to the operation occurred within the first four postoperative weeks, as was verified in a subsequent study.[21] The more serious complications which must share in the responsibility for the slow acceptance of the operation are trauma and infection leading to massive hemorrhage and/or bronchopleural fistula and mixed infection empyema, the chief contributors to death attributable to the operation.

In speaking of technical results of pneumonolysis, the advantages of the terms complete and partial over the terms technically successful and unsuccessful must be pointed out; the former are more descriptive and tell exactly what was accomplished, whereas the latter are open to various interpretations. The clinical results of pneumonolysis, a very popular expression in the literature, should be regarded as a misnomer because the only direct results of pneumonolysis are the complications attributable to the operation and the mechanical alterations in the character of the collapse; the clinical results are those of pneumothorax and not of pneumonolysis.

Standardization of terminology is urged so that comparison of results may be more meaningful; sputum conversion statistics following pneumonolysis, for example, mean little unless it is known that a contralateral or extrapulmonary source of positive sputum does not exist. Just as important are the time interval between operation and evaluation of results and the criteria of sputum conversion. It is probable that few sanatoriums were as particular regarding the last mentioned as those of Barlow[29] and Pottenger.[38] However, this circumstance probably does not detract from the value of the data gathered because in the majority of the reports the criteria were probably uniformly less exacting than those in the sanatoriums mentioned.

A gradual and steady trend toward both earlier pneumonolysis and broadening of the indications for the operation was noted in the literature reviewed. This changing attitude must be attributed to the recognized superiority of clinical results and lower incidence of complications in pneumothoraces initially free or surgically freed of restricting adhesions. If pneumothorax is at all indicated, a concentrically free collapse should be our aim and if restricting adhesions are present and thoracoscopy demonstrates that they can be released without risking severe complications pneumonolysis is indicated.

Only thoracoscopic examination can determine the character and feasibility of the adhesions and often it only can answer the question whether or not adhesions are present. Indications for thoracoscopy and consideration of pneumonolysis that I advocate are: (1) x-ray evidence of adhesions which are not obviously diffuse and (2) a persistently positive sputum with or without cavitation despite absence of x-ray evidence of adhesions.

The optimum time for thoracoscopy is one to two months following induction of pneumothorax provided the pneumothorax space is large enough to allow instrument manipulation. Adhesions found attached to the diseased lobe should be released if this can be done safely and without obvious risks. Only in specially desperate circumstances where no other collapse procedure is feasible is one justified in undertaking a pneumonolysis involving obvious risks of serious postoperative complications. Adhesions in the presence of patent cavitation or positive sputum arising in the parenchyma of the collapsed lung constitute a more urgent indication for pneumonolysis than adhesions over a controlled lesion in the symptom-free patient. The only contraindications to pneumonolysis are: mixed tuberculous empyema, acute febrile pleural effusion, serous or purulent and progressing obliterative pleuritis.

Since the publication of Moore's collected series in 1934, more than two and one-half times as many pneumonolyses were reported as during the preceding decade. Despite this increasing employment of the procedure in the improvement of induced pneumothorax, it must be admitted that thirty years after the introduction of closed pneumonolysis its acceptance by the profession is still incomplete. There still remain sanatoriums in which pneumothorax, phrenic nerve interruption, and thoracoplasty are performed regularly but no pneumonolysis. As long as pneumothorax retains its role of the most commonly practiced collapse procedure, thoracoscopy and pneumonolysis must be accepted as integral parts of such a collapse program both in order to improve the character of the collapse in those in whom it should be maintained and to eliminate those in whom pneumothorax is useless or even dangerous and in whom it should be abandoned in favor of a more effective procedure.

Assistance in tabulating by June Von Hofe is hereby gratefully acknowledged.

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SURGICAL MANAGEMENT OF RESIDUAL TUBERCULOUS CAVITIES FOLLOWING PRIMARY THORACOPLASTY

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IN THE treatment of pulmonary tuberculosis modern extrapleural thoracoplasty has no counterpart in properly selected cases. Throughout the past years there has been a progressive improvement in the selection of cases, in the surgical technique, and in the postoperative care and management, yet after the most exacting attention to detail in each of these departments, there remain a few cases in which a complete closure of the cavity has not been realized and the sputum continues to show the presence of tubercle bacilli. This is a problem that confronts any of us who are engaged in the surgical treatment of pulmonary tuberculosis. Alexander,[1] in the report of his results from secondary thoracoplasty, stated that 21.4 per cent of 14 patients still have positive sputum. Dolley, Jones, and Paxton[7] found 396 out of 1,636 cases with positive sputum due to incomplete approximation of cavity walls and active bronchial or tracheal ulceration. Recently O'Brien and his associates,[20] in reviewing 511 cases, stated that 12.98 per cent did not have cavities closed after operation.

Assuming that the surgical collapse is otherwise adequate as a result of the resection of the transverse processes, and sufficient lengths of the anterior portions of the ribs, the principal remaining procedure thus far accepted and generally recommended is the secondary or revision thoracoplasty, unless one of the less widely used procedures, to be discussed later, are undertaken.

The rate of incidence of this secondary operation can be reduced in several ways:

1. The resection of the transverse spinal processes and sufficient lengths of the necessary number of ribs at the time of the first series of operations
2. Judicious application of formalin to the periosteal beds at the time of the first stage
3. Elimination of undue delays between stages
4. The performance of an apicolysis (Semb procedure)[24]
5. Maintenance of an adequate period (seven months or more) of strict bed rest following the completion of the surgical program

Failure to resect the transverse processes (Fig. 1) contributes to the maintenance of the posterior vertebral gutter area into which cavities

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From the Surgical Clinic of the Hamburg State Sanatorium.

can "migrate" and thus minimize, in part at least, the effect of the surgical collapse. Similarly, too long anterior rib (Fig. 2) segments will prevent an adequate collapse of the underlying diseased lung tissue. Particularly is this true in the case of giant cavities and large underlying empyema spaces. In this clinic, it is the practice to resect all transverse processes, all of the first and second ribs, and generally all of rib three. Other ribs are resected as far anteriorly as each individual case demands. Long anterior segments are generally not permitted to remain. This practice has all but eliminated the anterolateral operation, but has not lowered our estimation of it as a valuable procedure when it does become indicated. It is, however, felt that the efficiency of the collapse is considerably enhanced by resecting the maximum rib lengths at the primary operation if by so doing there is no increase in the danger of postoperative complications.

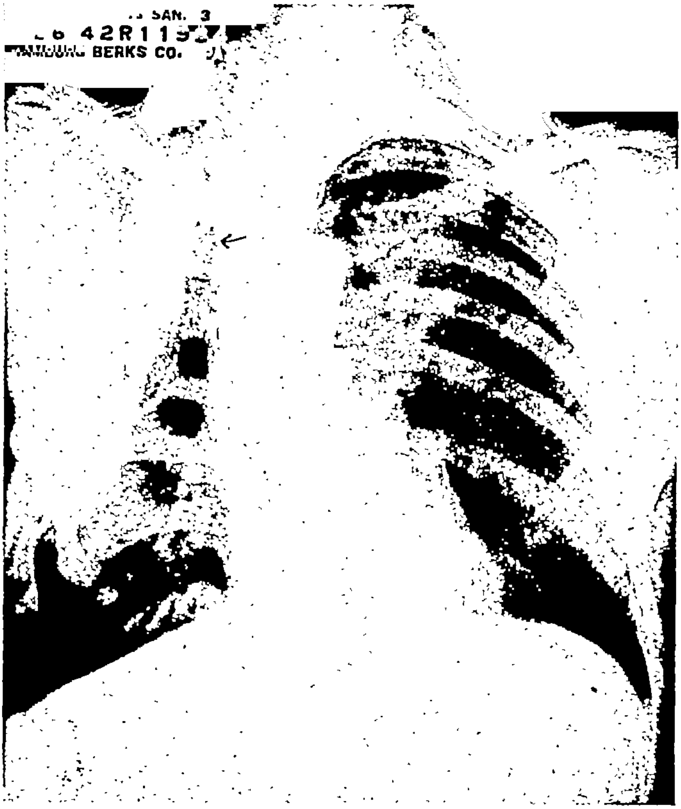


Fig. 1.—Tuberculous cavity still patent; inadequate resection of transverse processes.

Since the practice of applying 10 per cent formalin to the periosteal beds of the first three ribs, there has been little or no difficulty encountered as a result of reasonably prolonged periods of delay between the first and the subsequent stages. This is not true if undue delays occur, such as between the second and third stages. If such delays do occur, re-formed bone will have developed and must be removed if an effective surgical collapse is to be realized. The average optimum mean period between stages is estimated at about three weeks (twenty-one days).

The Semb procedure either as originally described,[24] or as described by Overholt[21, 22] has been essentially abandoned because of undue complications following this modification of the regular thoracoplasty procedure. These complications have been described previously.[11, 12, 14] I still feel that the violation of the integrity of the mediastinal lymphatics accounts in a large part for the undue rise in deep wound infections and that the Semb procedure, although of particular value in the closure of large and giant upper lung cavities, cannot be safely employed in these cases because of the possibility of disruption of an indispensable peripheral blood supply to the cavity wall. It is quite possible that certain of the large upper lobe cavities that remain partially uncollapsed following the usual thoracoplasty procedures (Figs. 3 and 4) would close if an extrapleural apicolysis had been performed, but I am not yet wholly convinced that the dangers do not outweigh the advantages in this type of case (Fig. 5).

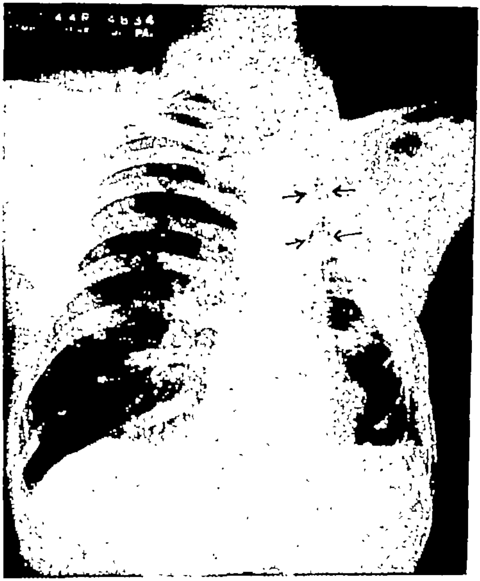


Fig. 2.—Inadequate resection of transverse processes and too long anterior rib stumps. Large cavity remains incompletely collapsed.

Maintenance of strict bed rest following completion of the surgical program is considered to be absolutely essential to the ultimate cure of the disease. Violation of that routine, among other things, will result in a higher percentage of "nonconverted sputum cases" and poor postoperative results, thus increasing the incidence of secondary operations. This latter operation will likewise frequently fail in the accomplishment of desired results if the program of strict bed rest and sanatorium regime is again violated. A minimum of seven months of such care with the use of wedge pillows, shot bags, etc., is strongly recommended.

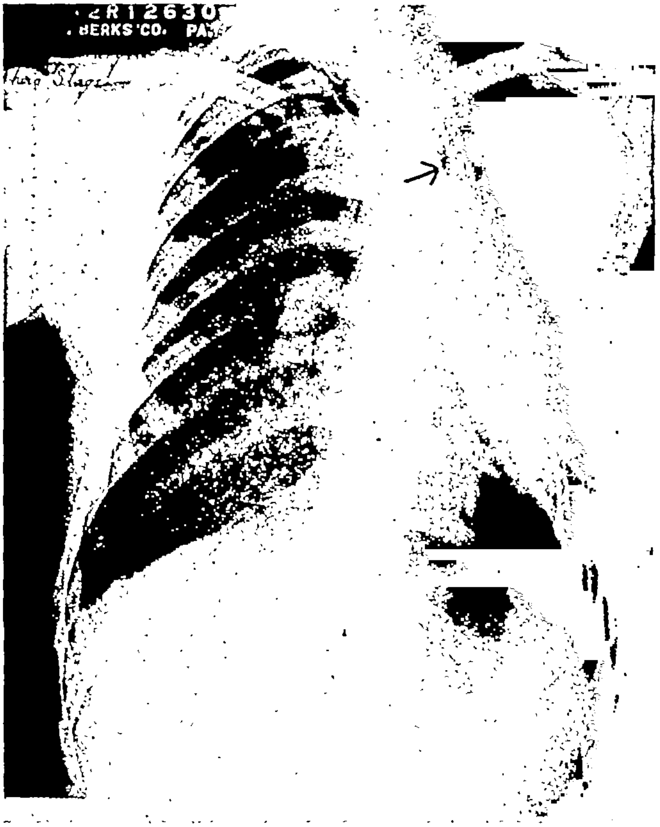


Fig. 3.—Incomplete collapse of cavity following "adequate" primary operation (thoracoplasty). Sputum is negative and patient is able to work following "mobilization and fixation" operation. (Roentgenogram of patient before "mobilization and fixation" operation.)

Early in this present series, we were confronted with the problem of a cavity which had actually ballooned out beneath the (Fig. 6) scapula and above the level of any re-formed bone (third rib periosteal bed and upward). It was for the control of this cavity that the technique hereinafter to be described was devised. In brief, this procedure consists of the mobilization of the lung and its suture or plication upon itself (Fig. 7, *A* and *B*). Since the first few cases, it has been adopted to a large extent as a part of most revision operations. Up to the time of this report 16 patients have been subjected to the modified procedure. Good results have been realized in all instances although the number is too small to permit of definite conclusions regarding the value of this or any operative technique; however, it is felt that a very distinct advantage has been gained.

The position of the cavity, the character of the cavity wall, and the presence or absence of an increased intracavitory pressure are considered to be important factors militating for or against the success of a well-performed primary operation. Of these factors the position of the cavity in the anteroposterior plane has heretofore been least discussed.

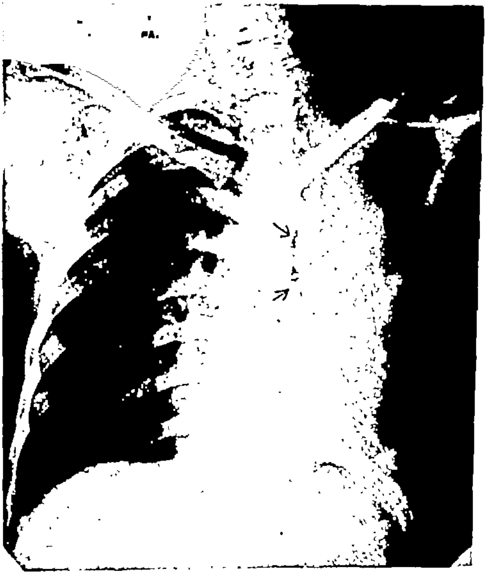


Fig. 4.—Residual cavity following primary thoracoplasty. Cavity is closed and sputum is negative following lung mobilization and fixation.

It has been observed that the greater majority of tuberculosis cavities (Fig. 10, *A* and *B*) when viewed on the roentgenogram following a lateral projection are situated posteriorly. This peculiarity explains for us one of the reasons why obliteration of the posterior vertebral gutter must be accomplished and maintained if the complete collapse and closure of many pulmonary excavations is to be realized. By the same token, it seems to explain why certain cavities persist in remaining patent not only following the primary but likewise following some secondary operations. It was reasoned, therefore, that if this anatomic situation could be altered these refractory cases could be effectively controlled and the dangers attendant upon the Semb procedure, and others, eliminated. The technical problem did not present any unusual difficulties although certain cautions had to be observed. These will be described later in this communication. The maneuver was carried out somewhat timidly at first, but is now considered to be well established without having incurred troublesome complications, and with sufficiently encouraging results to warrant its continuance in our clinic.

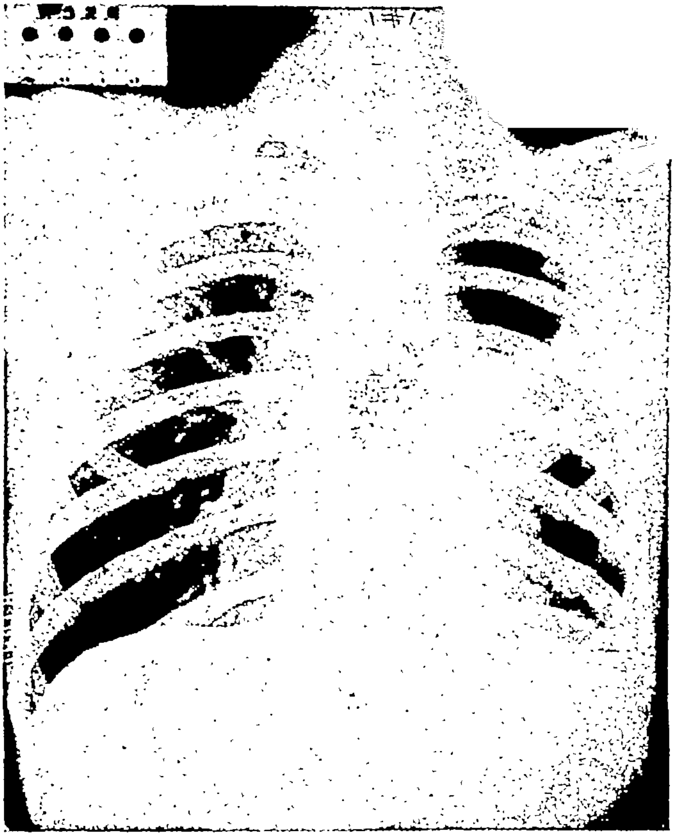


Fig. 5.—Multiple and multilocular cavitation of upper two-thirds of left lung; entire upper lobe excavated. Apicolysis is considered dangerous in this type of case.

Although this apparently new maneuver has aided tremendously in reducing our percentage of "nonconverted sputum cases" we always attempt to carry out the primary series of operations in such a way that a maximum mechanical collapse is realized. If then, cavity closure is not accomplished (Figs. 1 to 5) a secondary operation (revision) is undertaken and "mobilization and fixation" maneuver incorporated completely or partially in the majority of cases.

Before proceeding to a consideration of the technique, a short review of the salient anatomic features and their significance seems worth while.

Anatomy.—It is superfluous to describe all anatomic structures and relationships associated with the area involved in the operative field under consideration; however, certain anatomic features will bear special consideration. These include the external and internal intercostal muscles and some of the structures that are in relation with the bodies of the vertebra.

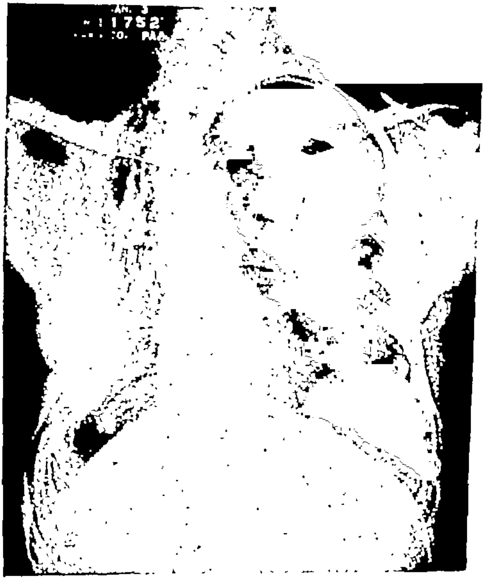


Fig. 6.—Showing the ballooning out of a large cavity after completion of a primary thoracoplasty. Complete closure and negative sputum resulted immediately after mobilization and fixation.

The external intercostal muscles have their origin at the costal tubercle posteriorly and continue anteriorly to the costochondral junction from whence they are continued to the sternum as the anterior intercostal membrane. The internal intercostal muscles, in contrast, continue from the sternum to the angles of the ribs as muscles, but from the angles of the ribs backward to the vertebra are continued as the intercostal membrane.

The intercostal vein-artery-nerve (intercostal bundle) lies between these structures and beneath the lower edge of the rib except from the angle of the rib backward, where they lie in the middle of the intercostal space. The arteries arise directly from the thoracic aorta which lies slightly to the left of the midline of the bodies of the vertebra. The veins drain into the azygos or hemiazygos veins directly or indirectly through the highest intercostal on the right or the accessory hemiazygos on the left side.

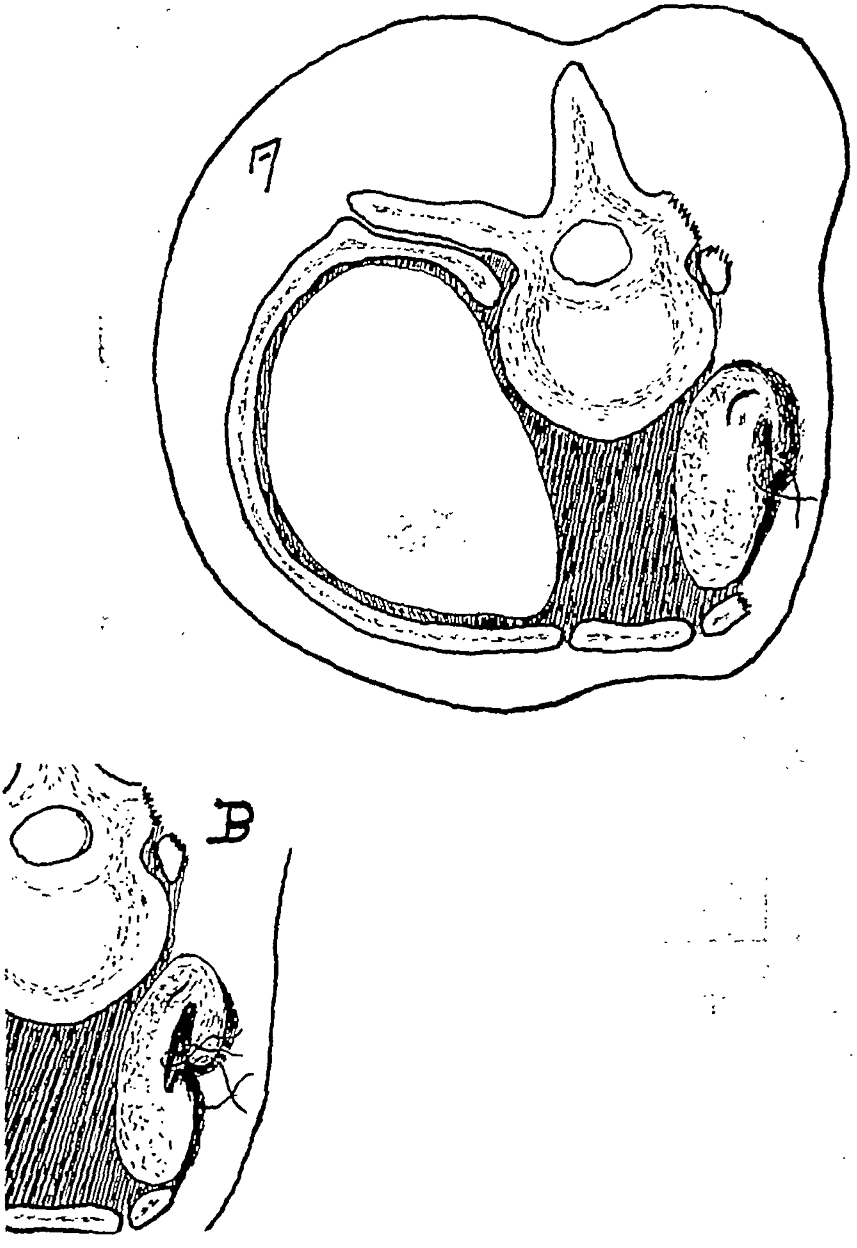


Fig. 7.—*A*, A schematic drawing showing the manner in which the lung is rolled forward and sutured (pleated) after mobilization posteriorly. *B* (insert), Suture fixation of the lung after mobilization both posteriorly and anteriorly. Anterior mobilization is more adaptable in the apical and subapical regions and in "small chested" patients.

On either lateral aspect of the vertebral bodies and ventral to the intercostal bundle lie the sympathetic chain. Coursing upward, in the midline, and lying directly upon the bodies of the vertebra is the thoracic duct.

The aorta, the esophagus, and other structures are present but as they are not too intimately related to the surgical maneuver under discussion nor subject to injury, they will not be described at this time.

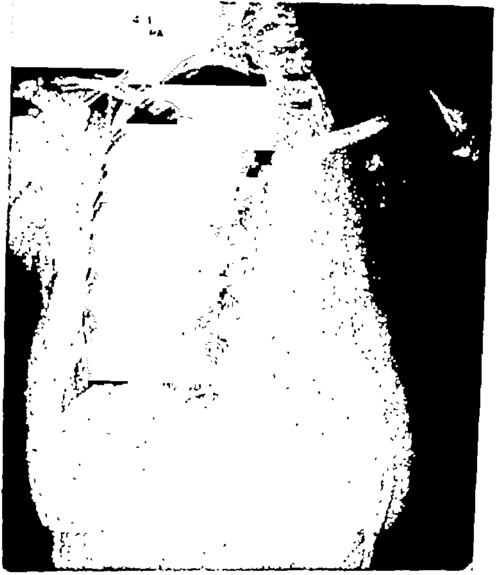


Fig. 8.—Results following lung mobilization and fixation and subtotal scapulectomy; 90 per cent of left lung originally contained multiple large cavities. Present status excellent, sputum negative eighteen months.

The significance of these features as they influence the surgical procedure is as follows: The external intercostal muscles may be divided posteriorly with complete impunity. The internal intercostal muscles do not have to be divided and if the intercostal membrane is sectioned the intercostal structures as a whole are carried along with the lung. The artery, vein, and nerve can be dissected free with ease for a considerable distance forward. Thus, collateral circulation to cavity walls will not be interrupted. Because of the overlapping and close proximity of one intercostal artery to the other, it is possible that one may be divided and yet an adequate blood supply will remain to preclude any necrosis of the cavity walls.

It is also noted that with careful dissection posteriorly there will occur no damage to the sympathetic chain although it is usually elevated and may be somewhat displaced when the whole maneuver is carried out in its entirety. Incidentally, a profound reflex response has been noted in the blood pressure (severe drop) and heart rate (marked increase, shallow volume) when the sympathetic chain is unduly manipulated. This emphasizes the need for gentle and careful handling of tissue during the dissection.

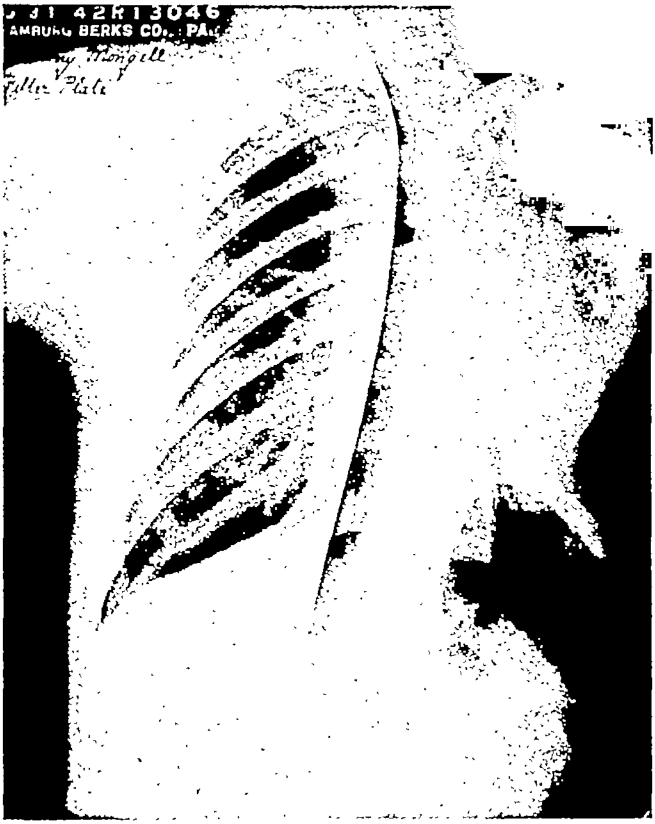


Fig. 9.—Cavity closed, sputum negative, results following lung mobilization and fixation. Same case as Fig. 3. Note area of aerated lung tissue preserved at base.

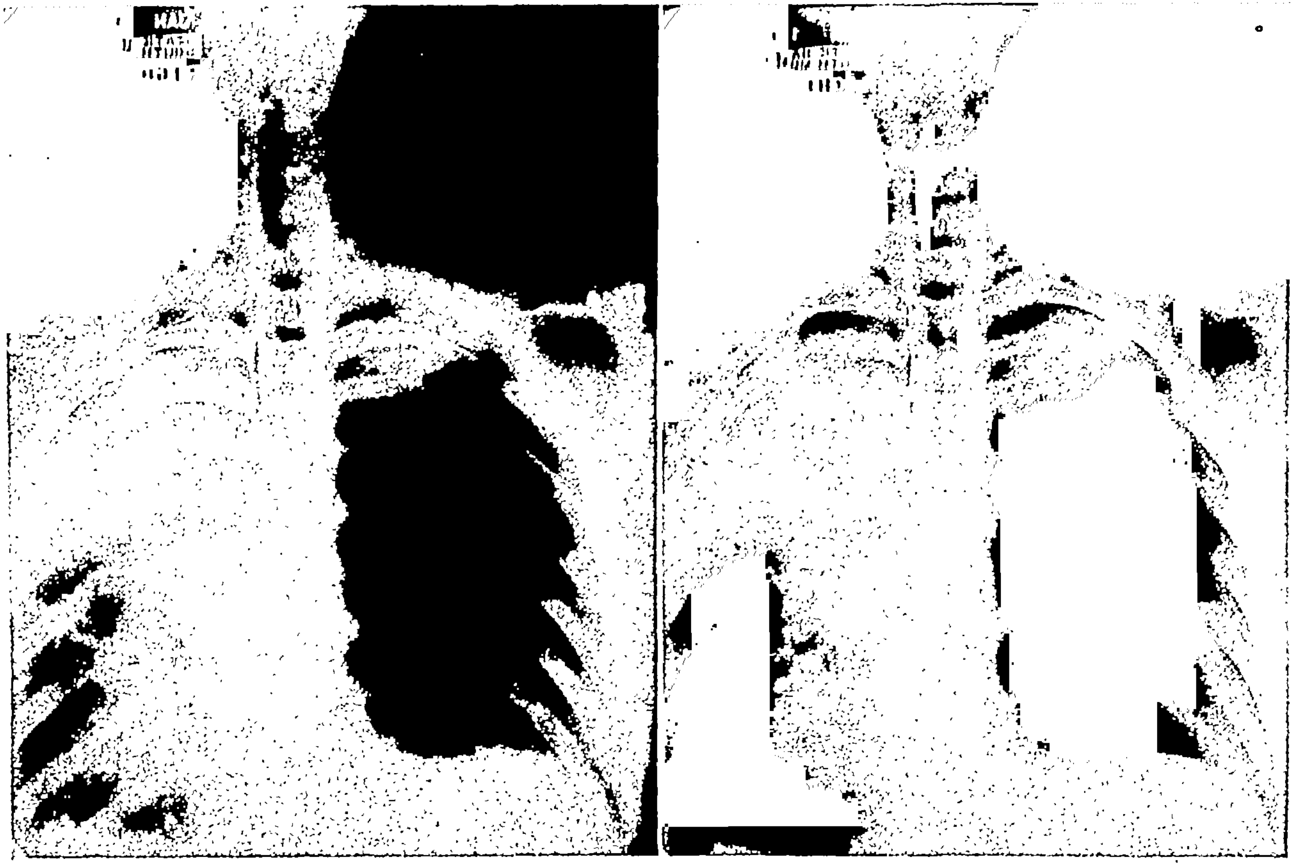
The positions of the aorta, thoracic duct, etc., are such that even with the most extreme dissection these structures will not be damaged or interfered with. Although on the left side the aorta is not infrequently palpated, with average care it is not at all subject to damage.

Technique.—The technique followed in removing the regenerated bone corresponds to that described by Alexander.[1] After this routine has been completed the lung is mobilized anteriorly by releasing fibrous and callous attachments around the anterior segments or sternal ends of the ribs. Portions of the anterior rib stumps are resected to whatever extent is possible or is deemed necessary for the particular case at hand. Next, *the scar tissue along the spine is incised longitudinally*, following a line overlying the previous sites of the transverse processes, except the first and usually the second. The line of incision is carried deep to the bony bed that was left after the resection of the transverse processes. From this point on, blunt dissection is employed as much as possible. The greatest of care must be exercised not to injure the underlying blood vessels and lung. Thence, a free cleavage plane is easily established by dividing the loose areolar attachments—the endothoracic fascia. The plane of dissection is developed so that the lung and pleurae are elevated out of the posterior vertebral gutter and from off the lateral aspects of the vertebral bodies. If this dissection is carried out bluntly and carefully a minimum of bleeding and damage to the intercostal structures will be encountered.

The intercostal bundles, especially the vessels, are dissected forward and their posterior portions freed as far as necessary without endangering any existing collateral blood supply to underlying lung tissue or cavity wall. The lateral and anterior aspects of the vertebra are approached. Care is exercised not to damage either the sympathetic trunk, the thoracic duct, or other pertinent anatomic structures. The extent to which the lung mass is elevated from off the bodies of the vertebra is tempered by the needs of each individual case. However, in all cases where this maneuver is undertaken, the lung is always elevated from out of the vertebral gutter and from off the posterior one-half to two-thirds of the lateral aspects of the bodies of the vertebra. *Posteriorly the dissection is carried upward to the second* dorsal vertebra and downward to the vertebral body of the last rib resected.

The lung, thus mobilized, is then rolled forward and sutured in position from above downward (Fig. 11). The sutures, black silk and No. 2 chromic catgut doubled, are passed through the dense fibrous tissue overlying the vertebral aspect of the lung mass (posteriorly) and through the similar fibrous mass anteriorly or through the cartilaginous chondrosternal portion of the resected ribs. The sutures are tied more snugly above and less snugly below. Caution is constantly exercised so that undue *constrictions will not be formed by the sutures* or possibly by cicatricial bands developing at a later time. There results from this procedure a complete collapse of the posterior and lateral portion of the lung although a portion of medial and mediasternal areas may remain aerated. The wound thereafter is closed in layers in the usual manner without drainage.

Indications and Contraindications.—The indications consist principally of the same indications as for any secondary or revision thoracoplasty. That is, the closure of tuberculous pulmonary cavities that have not been closed by an adequate primary thoracoplasty in a patient who is physiologically qualified to undergo an operation of this



A. *B.*

FIG. 10.—*A*, Laminograph 4 cm. from table top shows pulmonary cavity to the best advantage. Upper lung opacity is result of postpneumothorax pleural effusion. *B*, Laminograph of same patient 6 cm. from table top. This shows cavity "fading out." Posterior position of cavity is thus demonstrated.

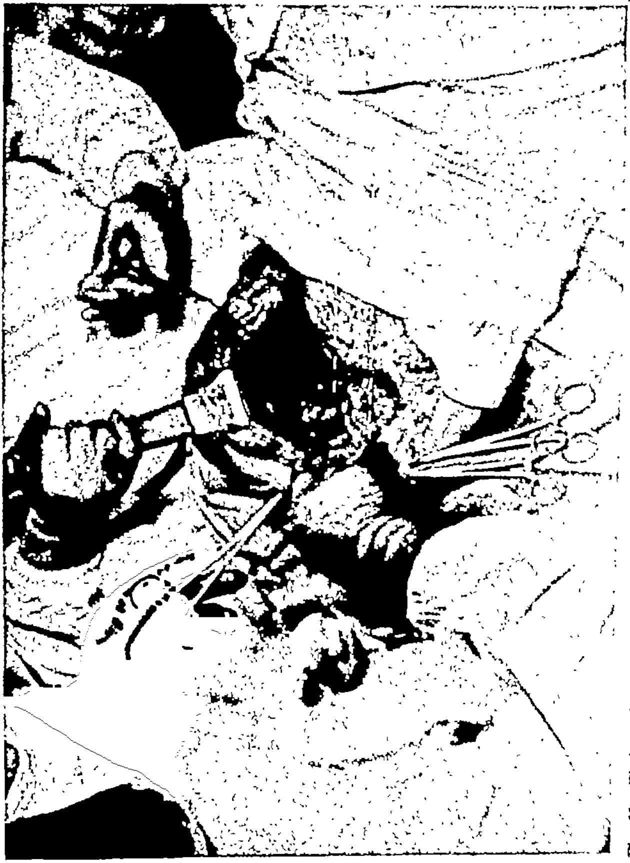


Fig. 11.—Photograph at completion of fixation operation. *a*, Undisturbed bony thorax; *b*, black silk and No. 2 chromic catgut sutures; *c*, tip of probe resting on body of vertebra.

magnitude. The uncollapsed cavity should be demonstrated on the routine roentgenogram or by a satisfactory bronchography.

It is absolutely contraindicated at the time of the primary operation, because of the inadvisability of passing sutures through the lung parenchyma and the limited scope of the operative field of each stage. Other contraindications include those for any thoracoplasty or any major operation. It is also inadvisable before an adequate interval of time has elapsed following the last primary thoracoplasty stage. It has been our practice not to proceed until the newly formed bone has become sufficiently well defined to render removal of it as easy as possible and further collapse of the diseased area following the primary operation. Eight to ten weeks are suggested as a minimal time limit following the last primary thoracoplasty stage. This time limit has the added advantage that the scar tissue will also have had a sufficient period of time to strengthen and develop and thus can support the heavy sutures that are to be required.

Up to the present time this procedure has not been employed in empyema cases. Complete resections of the first three ribs and long sections of the others has proved adequate to close all pure tuberculous empyema spaces and all mixed tuberculous empyema spaces usually down to the fifth or sixth rib. Below this level it has been necessary to employ the Schede procedure in the majority of cases, particularly if the empyema has existed as such for any length of time.

Pre- and Postoperative Care and Anesthesia.—Preoperative sedation is regulated according to the calculated amount of each individual patient. The average sedative, however, consists of morphine sulfate, gr. $\frac{1}{6}$, and scopolamine hydrobromide, gr. $\frac{1}{100}$, preceded by the intravenous administration of 1,000 c.c. of 5 per cent glucose. All patients are given postural drainage preoperatively. The anesthetic mixture is high in oxygen and the carbon dioxide absorption technique is employed, thus providing a warm moist anesthetic mixture.

Cyclopropane has been used exclusively as the anesthetic agent in all our thoracoplasty cases irrespective of the type of operation. Careful management of the depth of the anesthesia during the various phases of the operation minimizes the degree of paradoxical motion.

One factor which I consider of great importance is the patient's position on the table. Before induction the patient is placed in the desired position for operation and no marked change is permitted thereafter. Extensive manipulations of the patient after induction have often been found to lead to undesirable vascular and sometimes respiratory changes.

Immediately postoperative each patient routinely receives a 500 c.c. transfusion followed by 1,500 c.c. of 5 per cent glucose intravenously, which is administered at approximately 600 to 800 c.c. per hour. Oxygen by nasal mask is always used for a varying duration. Coughing is encouraged and opiates are administered for the control of pain but as sparingly as possible as a precaution against depression of the cough reflex. *As soon as tolerated, sandbags, shot bags, and wedge pillows* are used. Posture is regulated as an additional countermeasure directed against the development of scoliosis. However, since fixation by suture of the mobilized lung in the collapsed state is the fundamental principle of this surgical procedure, it is doubted that compression measures will add much to the collapse. Nevertheless, their use is being encouraged but not urged if their application proves too painful.

Complications.—Collection of various amounts of fluid within the wound area is the only complication thus far encountered. In one case the quantity of fluid was sufficient to cause some distention of the superficial soft tissues and some pain. In this case only needle aspiration was carried out. The amount of fluid removed was just sufficient to afford relief. Fluids have all been absorbed in one to two weeks without incident. The general policy has been to permit the fluid to remain and be absorbed spontaneously. No infections have occurred.

Although no other complications so far have been encountered, it is believed that care must be exercised during the suturing and fixation of the lung to arrange the sutures so that constricting bands will not be formed, otherwise adequate drainage of the lung will be interfered with and the several sequelae of such would naturally ensue. In our series it is too soon to have discovered the full series of complications that may develop. It is, however, remarkable to note the minimum of reaction shown by these patients to the operation, even though the operations and necessary trauma and blood loss are most severe.

Advantages.—I have come to feel that this procedure, which has been termed "lung mobilization with fixation," offers definite advantages over any procedure yet suggested for the closure of these refractory cavities although this too possesses certain limitations.

The danger of wound infection and necrosis of the cavity wall is eliminated due to the fact that a sufficient amount of the peripheral vascular supply to support the life of the tissues of the cavity wall remains uninterrupted and undisturbed. The mediastinal lymphatics are not disrupted and therefore deep wound infections that apparently result therefrom have not occurred.

There is created a definite "positive or forceful collapse of the cavity" whose walls may be resistant, or the cavity may be a "positive pressure cavity," or both. In either event the factors are overcome by the counterforce exerted when the lung mass is rolled forward, sutured, and maintained in that position. The relatively permanent suturing with black silk provides a means whereby the lung is held in position out of the posterior vertebral gutter, a position more favorable for the absolute closing of cavernous lesions.

Lung mobilization and fixation represents a procedure that may be used routinely with any revision thoracoplasty without incurring any appreciable added surgical shock or postoperative complications. A second revision operation at a later date is much less likely to be needed than if this procedure was not incorporated into the usual revision operation. Lastly, but far from least, is the tremendous reduction in the degree of paradoxical motion and the possibility of a "flail chest" postoperatively, a condition feared after some of these secondary or revision operations.

RESULTS

Within the past three years (1941-1944), 203 patients have come to thoracoplasty of all types for pulmonary tuberculosis or tuberculous empyema. This represents a total of 424 stages. In addition to these 203 patients, 39 have had 45 secondary (revision) operations. Twenty-three of the 39 (or 58.1 per cent) represent the "mobilization and fixation" type of operation.

At the time of this communication the sputum of all but 1 of the 23 (or 4.3 per cent) patients is free from tubercle bacilli. A few report 1 to 2 dr. of a mucowatery sputum daily. The remainder are cough- and sputum-free. Incidentally, it has been noted that these patients upon whom the details of the operation have been rigidly carried out have an early disappearance of both cough and sputum or at least a dramatic reduction in the amount accompanied also by a change in the character of the sputum; the change being from the purulent type to the mucowatery variety.

Of the remaining 16 cases, 3 patients still show tubercle bacilli (18.7 per cent) in their sputum although there is a good mechanical collapse. Every effort has been made to discover the cause of the bacilli in these cases but so far without avail in 2 cases while in 1 there was an extensive tuberculous bronchiectasis involving the lower portions of the lung. A fourth case was positive but the patient was operated very recently and although sputum is now bacilli-free, it is too soon to justify her inclusion in this series. (This patient has likewise had a mobilization and fixation operation.)

DISCUSSION

Among the many perplexing problems in the surgical treatment of pulmonary tuberculosis there is none more discouraging than the patient who continues to have tubercle bacilli in the sputum following a technically adequate thoracoplasty with a good resultant collapse of the lung. It may have been that the cavities never completely closed or once closed have reopened even after the lapse of many months. Why these cavities reopen or fail to collapse cannot always be determined. The "heavy walled cavity" and the "positive pressure cavity" are notoriously difficult to collapse; the resistant chronic fibrotic lung and the giant upper lobe excavations also present difficulties militating against the success of a correctly performed primary thoracoplasty.

Over a period of years a variety of recommendations and suggestions have been designed to combat this problem, yet it still remains as a challenge to the thoracic surgeon. Supplemental procedures have been designed. Semb[24] and Overholt[21, 22] suggested an apicolysis, Head[15] suggested the "myoplastic thoracoplasty," Dolley and Jones,[6, 7] Alexander,[2] and others[5] have suggested pneumonectomy and lobectomy in certain selected cases. Coryllos and Ornstein[5] have reported open drainage or cavities with pedicled muscle flaps being used to fill these cavities. I had one such successful case in a patient who died approximately two years later of bilateral renal tuberculosis. Extrapleural gauze packs, Monaldi intracavitory drainage, and the flap drainage of Rogers and his co-workers[23] are among other recommendations that have been described to aid in the collapsing of these residual excavations. Eloesser,[9, 10] quoting Adams, suggests that some method of occluding the affected lobe will be devised. It appears from the results already obtained, that lung mobilization with fixation is potentially capable of accomplishing this aim.

In developing this procedure an effort has been made to devise a method that would sacrifice as little lung tissue as possible, entail a minimum of unfavorable postoperative sequelae, and subject the patient to a convalescence no more troublesome than the average secondary operation and yet fully obtain the goal of cavity closure.

The advisability of lobectomy and pneumonectomy is open to considerable question. The infrequency of proper indications and the inevitably high mortality as yet does not justify lobectomy or pneumonectomy in the majority of cases.

Such procedures as the open drainage of cavities, by Monaldi drainage[18] or by pedicled muscle flaps, violate a long-established principle, that of incising directly into a tuberculous lung with the consequent tuberculous sinuses and fistulas. The wound infections and postoperative atelectasis that are common in apicolysis and its several variations place special limitations on this maneuver. These have been absent in our mobilization and fixation cases, without incurring undue limitations in the flexibility of the operation. So far, we have not been called upon to carry our dissections below the level of the eighth dorsal vertebra. We are therefore not able, at this time, to comment on the possible difficulties or results involving the area below this level. Above this level the area involved in the procedure is determined by roentgenogram (with or without a lipiodol bronchography). Although the entire lung above the eighth rib cannot be wholly occluded, especially in individuals with large chests, the amount occluded can be regulated almost at will so that apparently no aerated lung tissue will remain if such is desired (Figs. 8 and 9).

The series of cases upon which this report is based is yet too small to justify definite conclusions. The possibility that the series will be rapidly expanded to satisfactory proportions is remote, for in any clinic the total number of revision or secondary operations is not very great. A report at this time is therefore considered justified so that others may accord this procedure a fair trial and the accumulated results therefrom will throw greater light upon the possible value of lung mobilization and fixation.

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A PNEUMONOLYSIS SPONGE CARRIER AND DISSECTOR

JAMES S. CONANT, M.D.
GLENN DALE, MD.

PRESENT instruments used for sponging the operative field during closed intrapleural pneumonolysis include metal applicators upon which cotton is cemented and bronchoscopic sponge carriers. Cotton applicators often foul the cannula, leave fibers of cotton within the chest, and the cotton occasionally drops off. Bronchoscopic sponge carriers are very lightly constructed and can carry only an exceedingly small sponge for intrathoracic work. They bend readily, especially when the operation proceeds to an awkward position of the chest, and do not always permit adequate pressure to be applied to bleeding points. Specially designed carriers are expensive and may break. For these reasons many operators prefer an olive tip laryngeal suction tube to keep the field clean and care little for mechanical wiping.

There are several advantages, however, in using sponges within the chest. They may be applied more gently and with better control than a suction tube, they afford traction to some degree, and are of considerable value in the enucleation of adhesions after the endothoracic fascial plane has been infiltrated with novocain solution, as originated by Ralph C. Matson, and the parietal pleura incised.[1]

For these reasons a new type of sponge carrier was developed and has been used intensively by the writer during the past year.* It consists of a 12-inch stainless steel rod, 5/32 inch in diameter with a handle having a recessed prong at its distal end. The flattened tip of the rod is 3/16 inch wide and contains a guarded prong within an elongated eye. The fabric used is gauze or bias tape ¼ inch wide.

The tape is first threaded through the eye of the carrier, then carried over the tip and pulled tightly as it is fixed on the prong at the handle. Moderate tension is all that is necessary to prevent the gauze slipping sidewise on the applicator tip. The tape is trimmed ¼ inch from the eye and the other end cut 8 or 10 inches beyond the handle. When blood has accumulated on the material at the tip the tape is merely released from the handle and drawn through the eye until a clean area appears, whereupon the tape is again fixed at the eye, trimmed, and fixed at the handle. This practice saves the time of rethreading. Time is also saved by having several of these instruments already threaded before the operation begins.

Because of temporary priority difficulties in obtaining bias tape, 1-inch gauze bandage was folded lengthwise twice by inverting the edges, and stitched slightly off center on a sewing machine. This material has no

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From Glenn Dale Sanatorium, Health Department, District of Columbia.

Received for publication Feb. 14, 1944.

*May be obtained from American Cystoscope Makers, Inc., Lafayette Ave., Bronx, New York, N. Y.

ragged edges, appears to be more absorbent than commercial bias tape, and does not interfere with the rapid insertion or withdrawal of the carrier from the cannula.

The instrument can be rapidly threaded in the dark by the nurse after a little practice. There is no danger of a pledget being lost. The instrument will withstand considerable pressure to control oozing, especially when work is done under adverse conditions. In enucleation it wipes the chest wall clean and does not shred fibrous tissue as does a cold cautery tip. Shredding gives rise to considerable oozing which obscures the field and frequently causes the surgeon to stop in the middle of an operation on a surface-to-surface attachment because of the fear that he may have lost the proper plane of dissection.

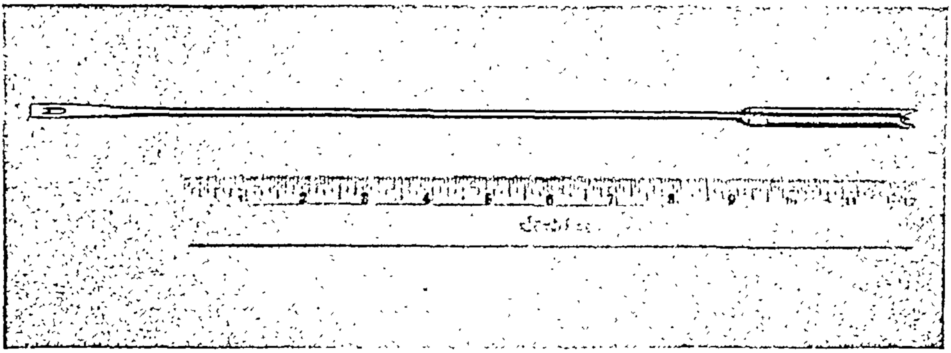

Fig. 1.

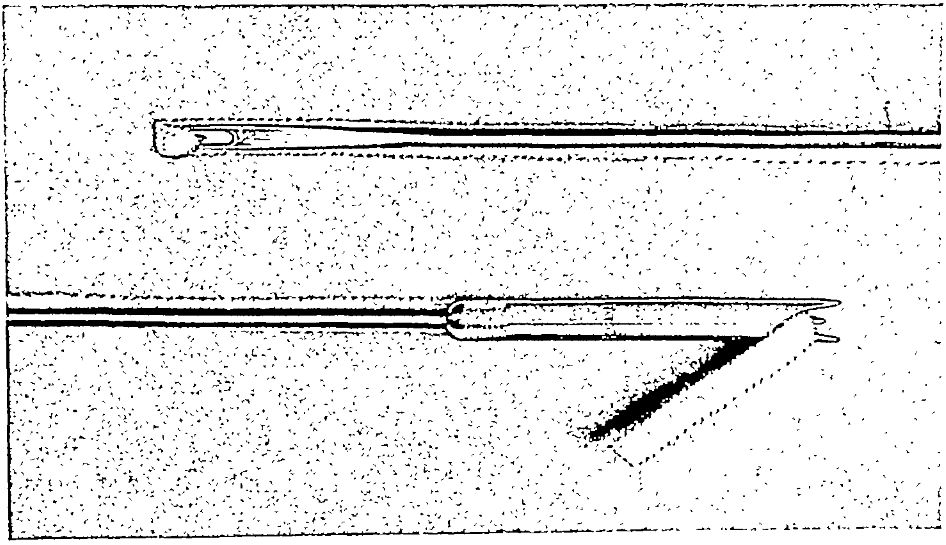

Fig. 2.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Twenty-Fifth Annual Meeting

THE twenty-fifth annual meeting of The American Association for Thoracic Surgery was held in Chicago at the Drake Hotel, May 5 and 6. In the absence of the president, Dr. Edward D. Churchill, who is a Colonel in the Medical Corps, Army U. S., overseas, the office was declared vacant and Dr. Frank S. Dolley, the vice-president, was elected to fill it. Dr. Claude S. Beck was elected to the office of vice-president. Both offices were thus filled for the duration of the meeting.

Colonel William L. Keller was elected to honorary membership, honoris causa.

The resignations of: Dr. John Powers, Dr. William H. Stewart, Dr. William P. Herbert, and Dr. Edward W. Peterson were accepted with regret.

The following active members were retired at their own request to senior membership:

Dr. Carl Eggers
Dr. William H. Thearle
Dr. Carl Steinke

The following associate members were elected to active membership:

Dr. Edgar Davis
Dr. H. McLeod Riggins
Dr. Herbert C. Maier
Dr. Brian Blades
Dr. John Garlock
Dr. Frederick Kergin
Dr. Arthur M. Vineberg
Dr. Michael DeBakey
Dr. Lyman Brewer
Dr. Paul Holinger

The following candidates were elected to associate membership:

Dr. Theodore L. Badger, 264 Beacon St., Boston, Mass.
Dr. O. Theron Clagett, The Mayo Clinic, Rochester, Minn.
Dr. Frederick D. Ackerman, 1374 Sherbrooke St. W., Montreal, P. Q., Canada
Dr. Herbert D. Adams, 605 Commonwealth Ave., Boston, Mass.
Dr. Howard Thomas Barkley, 1215 Walker Ave., Houston, Texas
Dr. Joseph Gordon, N. Y., State Tuberculosis Hospital, Bay Brook, N. Y.
Dr. Archibald J. Grace, 455 Waterloo St., London, Ontario, Canada
Dr. John Benson Grow, Fitzsimons General Hospital, Denver, Colo.
Dr. Cranston William Holman, 65 East 60th St., New York, N. Y.
Dr. Julian Johnson, 3400 Spruce St., Philadelphia, Pa.
Dr. Hiram Thomas Langston, 240 East Delaware Pl., Chicago, Ill.
Dr. Robert Carlisle Major, 384 Peachtree St., N.E., Atlanta, Ga.
Dr. Ross Robertson, Muskoka Hospital, Gravenhurst, Ontario, Canada
Dr. Dudley E. Ross, 974 Dunsmuir Rd., Town of Mount Royal, P. Q., Canada
Dr. Otto C. Brantigan, 104 W. Madison St., Baltimore, Md.
Dr. Dwight E. Harken, 171 Bay State Road, Boston, Mass.
Dr. Paul V. O'Rourke, 307 David Whitney Bldg., Detroit, Mich.
Dr. J. Ross Veal, Gallinger Municipal Hospital, Washington, D. C.
Dr. David H. Waterman, 607 Medical Arts Building, Knoxville, Tenn.

The by-laws were amended to delete Sections 4 and 10 of Article VII, thus making it unnecessary for senior members to pay dues.

The deaths of six members had occurred since the last meeting in 1941. They were:

Warren G. Breidenbach
Herman Fischer
Lincoln Fisher
Maurice Berck
William Branower
H. Kennon Dunham

Dr. Richmond L. Moore was appointed chairman of the Rose Lampert Graff Prize Committee, to succeed Dr. Frank Berry, now overseas.

The following men were elected to hold office until after the next meeting:

President, Dr. Claude S. Beck, Cleveland, Ohio
Vice-President, Dr. I. A. Bigger, Richmond, Va.
Secretary, Dr. R. H. Meade, Jr., Memphis, Tenn.
Treasurer, Dr. William E. Adams, Chicago, Ill.
Editor, Dr. Evarts A. Graham, St. Louis, Mo.

The following members were elected to the council: Dr. Frank S. Dolley to succeed Dr. Frazier Gurd as the retiring president for the one-year term of office, and Dr. Alfred Blalock to succeed Dr. Frank S. Dolley for the four-year term.

The time and place of the next meeting is to be decided by the new officers and council.

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ADJUVANTS WORTH CONSIDERING

Mayer—**Radiation and Climatic Therapy of Chronic Pulmonary Diseases**

Edited by EDGAR MAYER, M.D., F.A.C.P., F.A.C.C.P. *Assistant Professor of Clinical Medicine, Cornell University Medical College; Attending Physician, New York and Memorial Hospitals. Special Pulmonary Consultant, New York Department of Labor. First edition, 1944, 400 pages, 46 illustrations, $5.00.*

Thoracic surgeons, internists, general practitioners, chest specialists, radiologists, physiotherapists and all concerned with the treatment of chronic pulmonary diseases and all forms of tuberculosis will highly appreciate this very practical volume. To his own wide and varied knowledge and experience Dr. Mayer has been able to add that of twenty-two other very able contributors, so that the field is well covered from all angles and thoroughly up-to-date as to the therapeutic potency of the methods described for the conditions indicated. The five divisions of the book are as follows: **Light Therapy—Artificial Light Therapy—Solar Radiation and Climatotherapy—X-Ray Therapy—Surgical vs. Conservative Therapy.** The twenty-two contributors to this concise useful guide include besides the editor:

Louis B. Baldwin (Phoenix)
Irvin I. Balensweig (Cornell)
Alfred L. Briskman (Colorado)
William Chang (New York)
Anthony C. Cipollaro (Columbia)
William W. Coblentz (Wash. D. C.)
Lloyd F. Craver (Cornell)
Martin Dworkin (New York)
Earl C. Elkins (Mayo Clinic)
John N. Hayes (Gabriels)
Ira I. Kaplan (Bellevue)
H. Haig Kasabach (Columbia)
Eugene Kisch (New York)
Frank H. Krusen (Mayo Clinic)
Henry Laurens (Tulane)
Maurice Lenz (Columbia)
Horace LoGrasso (Perrysburg)
Harriet C. McIntosh (New York)
Clarence A. Mills (Cincinnati)
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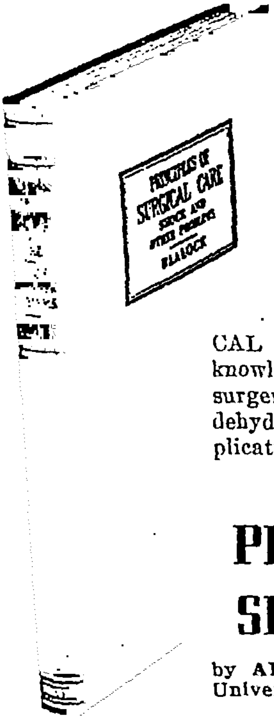

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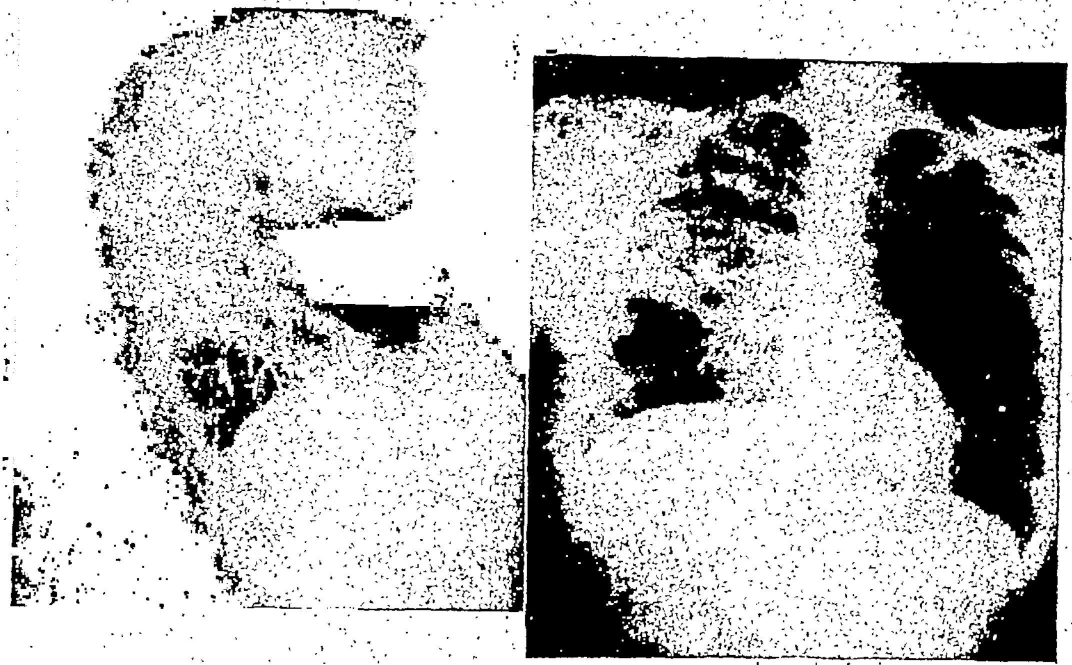

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And there's a day coming when every mother's son of us will want to stand up and yell, to cheer ourselves hoarse over the greatest victory in history.

But let's not start the cheering yet.

In fact, let's not start it at all—over here. Let's leave it to the fellows who are *doing* the job—the only fellows who will *know* when it's done—to begin the celebrating.

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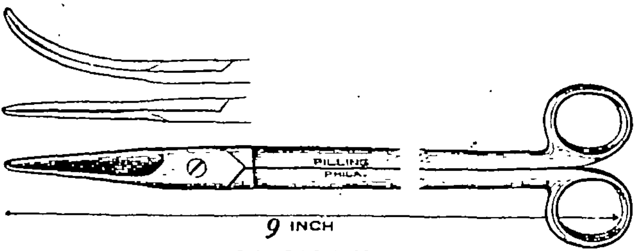

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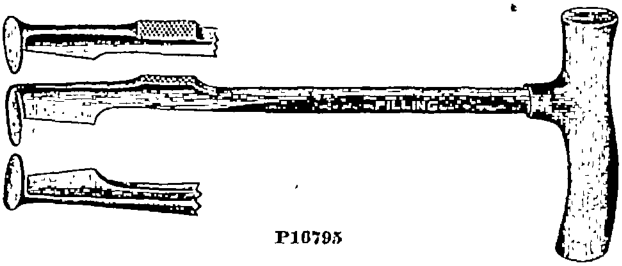

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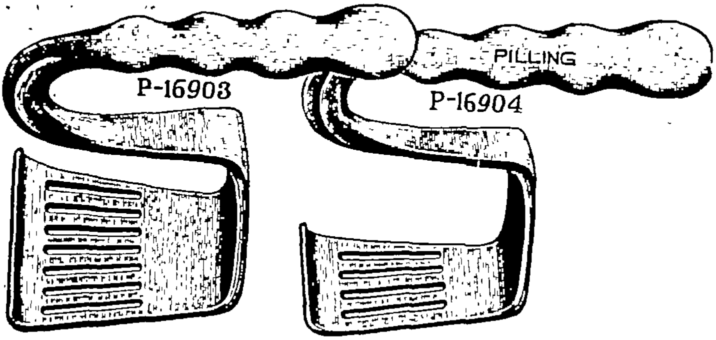

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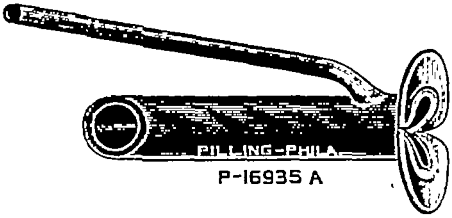

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The Journal of
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Vol. 13 October, 1944 No. 5

Original Communications

ARTERIOVENOUS FISTULA OF THE LUNG

A Report of a Patient Cured by Pneumonectomy

John C. Jones, M.D., and
(by invitation) William Paul Thompson, M.D.
Los Angeles, Calif.

Arteriovenous fistula of the lung usually masquerades in a *cyanotic young patient with clubbing of fingers and toes* as congenital heart disease or polycythemia until either a tumor of the lung or a continuous murmur in the chest is finally discovered. This report is concerned with such a case.

Four cases of arteriovenous fistula of the lung have been reported by Rodes,[1] Smith and Horton,[2] Hepburn and Dauphinee,[3] and Goldman.[4] These have all been summarized in Goldman's article, in which he reported his case in detail. Hepburn and Dauphinee's patient had a successful pneumonectomy by Shenstone and Janes and a diagnosis of cavernous angioma was made on the removed lung. Subsequently, Janes[5] treated bilateral multiple cavernous hemangiomas of the lungs by local resection of the tumors. The patient made a rapid recovery. Our case, so far as we know, is the sixth to be reported and the third in which operation was successful.

CASE REPORT

J. P., aged 24 years, a single, white woman, was referred, March 15, 1943, by Dr. Ralph F. Palmer of Phoenix, Ariz., to one of us (J. C. J.) for resection of a tumor of the right lung.

History.—The patient was cyanotic from birth and intensely so until the age of 16; thereafter, she is believed to have become somewhat less cyanotic. At birth and until the age of sixteen years, a soft, blowing,

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Read at the Twenty-Fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

basal systolic murmur without transmission was heard. Clubbing of the fingers began at the age of 9 years and progressively increased until 16, then remained stationary. She was restricted until that time, but was subsequently quite active without symptoms. One and one-half years before coming to us she was examined by an internist, and for the first time, an intrathoracic mass was seen by roentgenogram. On a presumptive diagnosis of tumor a course of radiation therapy was given, but it affected neither the mass nor the cyanosis. At that time polycythemia was noted and thought to be primary. The red blood cell count had ranged between 6.5 and 7.5 millions, with a hemoglobin of 130 per cent, for the last one and one-half years. There had been no abnormal white cells and there was no response to venesection done some time ago. She had been working steadily as a stenographer and leading a normal life.

Physical Examination.—The patient was well developed and nourished, of a slender build. She looked well and was a very handsome young woman. The hair, skin, and eyes were normal. The conjunctivae and mucous membranes were intensely cyanotic. The carotid pulsations were forceful and the jugular veins were not distended. The thorax was average in size and symmetrical. The heart was normal in size, with a moderate apex impulse and no thrill. There was slight sinus arrhythmia at a rate of 96 per minute. The sounds were normal with no accentuation of P_2. Murmurs were not heard. The lungs were clear. Over the right breast and just beneath it there was a moderately loud, rough, roaring murmur, probably continuous, of maximal intensity at the end of systole and the beginning of diastole. It was loudest at the end of inspiration and almost disappeared on expiration. There was no thrill. The pulses were normal. The blood pressure in the right arm was 112/94, in the left arm 110/92. The abdomen was negative. There was marked clubbing of the fingers and toes.

Fluoroscopy.—The right hilum was increased in density and a pulsating vessel radiated from the right upper hilum to a round mass (8 or 10 cm. diameter) just above the right diaphragm anteriorly.

Electrocardiogram showed a tendency to right axis deviation.

Urinalysis, blood Wassermann and Kahn tests were negative, and there was no sputum.

Hematology (as reported by Dr. Madeleine Fallon).—Hemoglobin 130 per cent; red cell count, 7,080,000; white cell count, 7,350. Differential: Neutrophiles, 63 per cent, lymphocytes, 25 per cent; monocytes, 12 per cent. Platelets were normal. The normal leucocyte count, the normal number of platelets, and the absence of changes in the blood, such as basophilic stippling and immaturity in the white cells, indicated that this was a secondary and symptomatic polycythemia.

A provisional diagnosis of arteriovenous aneurysm of the right lung was made on the history of cyanosis since birth, the persistent secondary or symptomatic polycythemia, the presence of a continuous murmur over the tumor mass in the right lung, clubbing of the fingers, and a normal heart. She was advised to return for hospitalization and further study.

April 8, 1943, she was admitted to the Hospital of the Good Samaritan and further roentgen studies were made. Roentgenograms and planigrams were interpreted by Dr. Richard Taylor (Figs. 1, 2*A*, 2*B*, 2*C*, 2*D*, and 2*E*). A series of body section radiographs was made from 3 to 16 cm. through the right lung. These demonstrate that the vessels to the right upper and to at least part of the right lower lobe were separate from those entering the large abnormal masses noted in the plain radiographs made elsewhere (Fig. 1). These masses appeared to be vascular in character and there was some indication that they were comprised largely of dilated branches of the pulmonary artery going to the right middle lobe. In the more anterior radiographs, a calcification made its appearance in the rounded lower mass. The mass appeared to be separate from the diaphragm. Fluoroscopically, the whole thing seemed to pulsate. Roentgenograms of the fingers revealed no bony changes. Fearing the dangers of thrombosis in patients with polycythemia we did not inject diodrast intravenously.

Further studies revealed the venous pressure to be 6 cm. of water. The plasma volume was normal but the whole blood volume was 170 per cent of normal.

Because the continuous murmur over the mass was greatly increased on full inspiration and almost disappeared on full expiration, it appeared that it might be of some clinical value to institute a right pneumothorax in an attempt to collapse the arteriovenous communication and maintain it for several months with the expectation that there might be some change in the cyanosis, polycythemia, and blood volume. Pneumothorax was begun April 12, 1943, (Figs. 3 and 4) and the patient was discharged home one week later to continue with pneumothorax under the care of her physician.

Sept. 18, 1943, she returned to the hospital after five months of pneumothorax therapy. Because of dyspnea and mediastinal herniation it had been impossible to produce sufficient intrapleural pressure to collapse the fistula completely. There was no change in the blood counts, blood volume, or cyanosis. It was of interest that she had noted transverse ridges on the fingernails and toenails which had appeared coincident with each pneumothorax refill. It appeared clear that an arteriovenous fistula of the right lung was present and that, despite failure of the mass to collapse completely, surgery was indicated.

Operation, September 20, 1943 (J. C. J.).— Under cyclopropane anesthesia by Dr. Lawrence Lee, a curved inframammary incision was

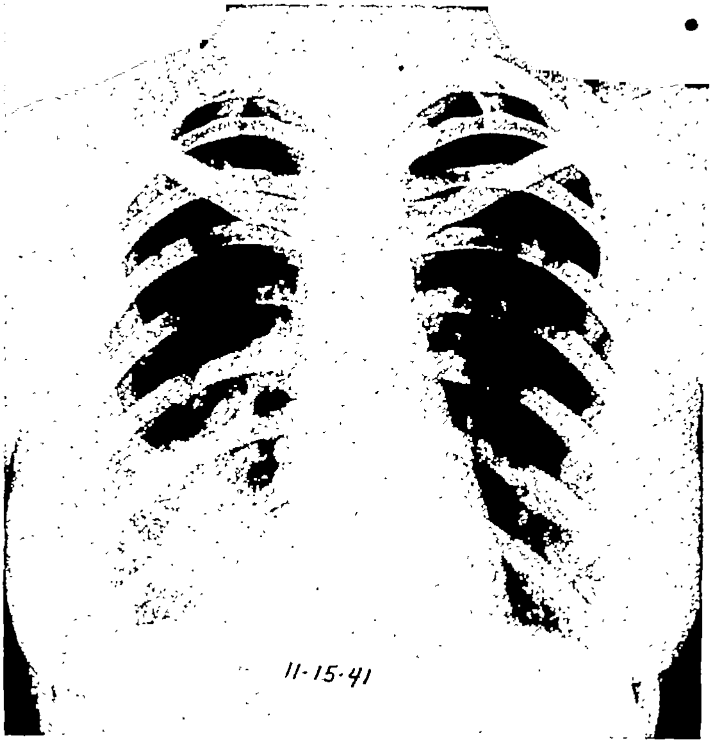

Fig. 1.—Anteroposterior roentgenogram revealing a multilobulated tumor extending downward from the right hilum.

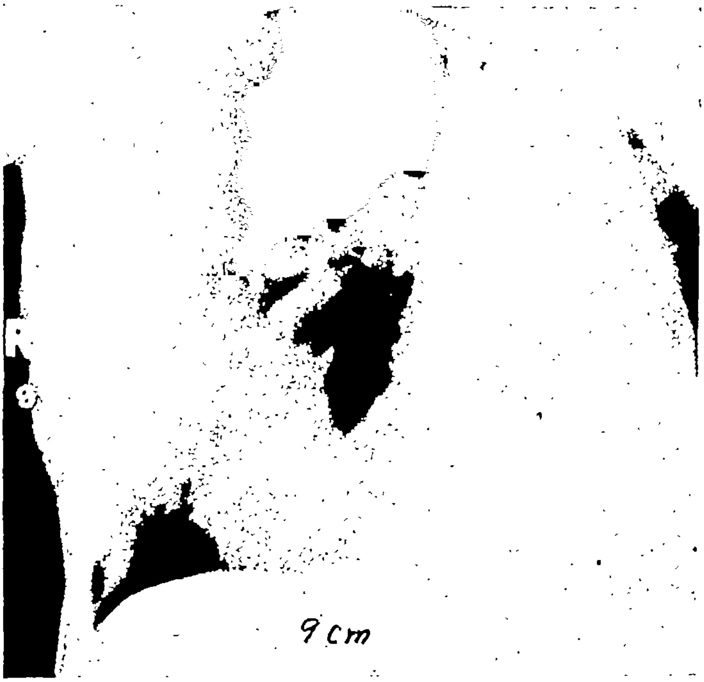

Fig. 2A.

Figs. 2A, 2B, 2C, 2D, 2E.—Planigrams at 9, 10, 12, 13, and 15 cm. levels, respectively, demonstrate a multilobulated tumor mass extending from branches of the right pulmonary artery. The more anterior radiographs (D and E) reveal an irregular calcific deposit in the lower rounded mass.

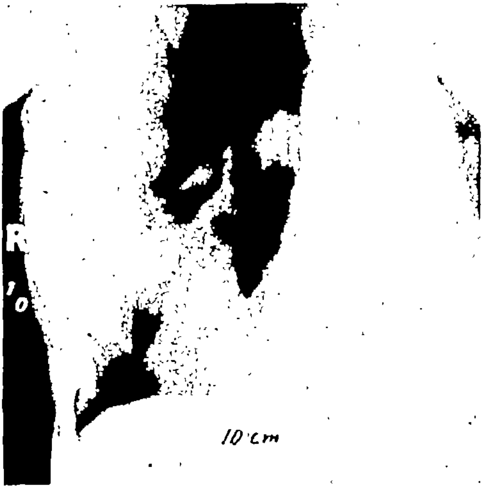

Fig. 2B.

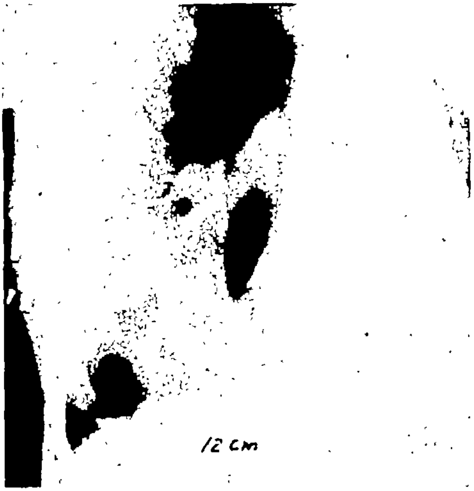

Fig. 2C.

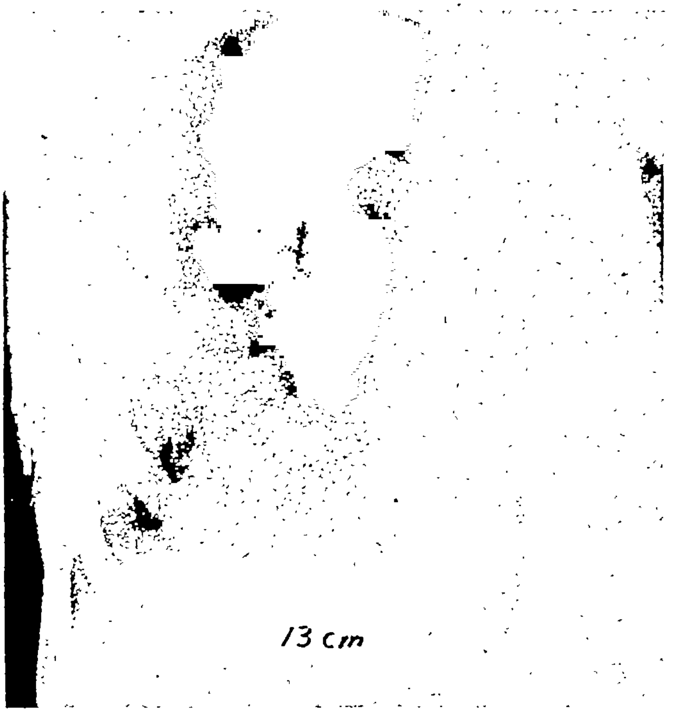

Fig. 2*D*.

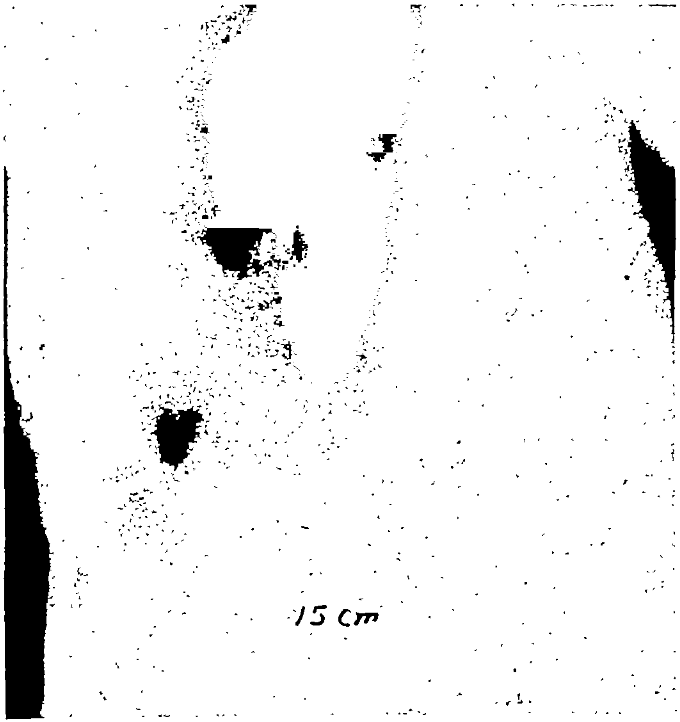

Fig. 2*E*.

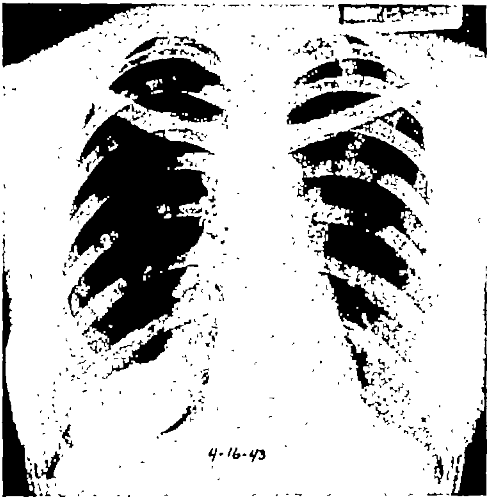

Fig. 3.

Figs. 3 and 4.—Posteroanterior and oblique roentgenograms after establishing right pneumothorax accentuate the lower tumor mass in the anterior position with apparent disappearance or collapse of other portions of the mass.

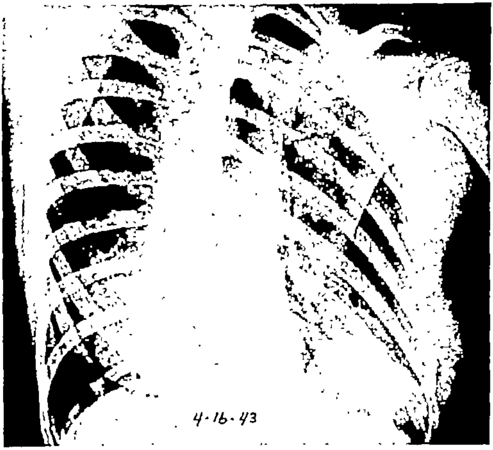

Fig. 4.

made extending from the third intercostal space anteriorly, under the right breast and upward and laterally to the third rib in the midaxilla. The pectoral muscles and breast were reflected upward and the pleural cavity opened by incising through the third intercostal muscles and pleura from the cartilages anteriorly to the posterior axillary line. Rib spreaders were placed and the ribs spread widely without resection or fracture. The right lung was 60 per cent collapsed by pre-existing pneumothorax. There was no semblance of a middle lobe and no adhesions. Over the lower half of the anterior surface of the upper lobe was a large number of blue distended varicosities. Attached to and suspended from the anterior and inferior aspects of the upper lobe (roughly corresponding to where the middle lobe should normally be located) a structure much the same as a distended gall bladder would hang from the liver, a reddish-blue pulsating mass the size of a lemon with a paper-thin wall, was seen to rest freely on the diaphragm. The right lower lobe appeared normal. What appeared to be the right pulmonary artery was large and dilated (3.5 cm. diameter). No superior pulmonary vein was seen, either at operation or in the specimen afterward. The inferior pulmonary vein was normal. Linen tapes were passed around the pulmonary artery and the inferior pulmonary vein. When the pulmonary artery was occluded by the tape, the pulsation in the tumor mass in the right upper lobe ceased, but there was no great change in its size. On occluding the inferior pulmonary vein the pulsation in the mass continued, the mass became larger and more distended, and the superficial varicosities over the surface of the upper lobe likewise became more prominent. It was obvious then, that the tumor mass had a direct communication from the pulmonary artery to the pulmonary vein and that since the inferior pulmonary vein emptied this vascular sac it was necessary to sacrifice the entire right lung. A right total pneumonectomy was done by dissection and individual ligation; first, the pulmonary artery was cut between transfixion sutures, then the inferior pulmonary vein likewise dealt with; finally the lung was amputated distal to a clamp applied to the right main bronchus. The right main bronchus was closed with eight over-end single stainless steel, 35 gauge wire sutures tied after the clamp was removed. The mediastinal pleura was closed after thorough irrigation of the stump and the pleural cavity with saline solution and after 5 Gm. of sulfanilamide crystals were sprinkled over the bronchial stump. The ribs were brought together with pericostal sutures and the chest wall was closed in layers without drainage. No blood transfusion was given because of the polycythemia, but a slow venoclysis of physiologic saline (1,000 c.c.) was administered during the surgery. She left the operation table in excellent condition.

Postoperative Course.—It was amazing to see the disappearance of the cyanosis in a matter of a few hours. She was given oxygen by

TABLE I

DATE	HB. (%)	R.B.C.	W.B.C.
3/15/43	130	7.08	7,350
9/19/43	130	7.11	5,700
9/20/43	104	5.90	(P.M. of surg.)
9/22/43	85	5.20	(2nd P.O. day)
9/24/43	73	4.10	(4th P.O. day)
10/ 8/43	86	5.30	(18th P.O. day)
1/10/44	80	4.04	9,350

nasal catheter for four days (routine procedure) although at no time was she *cyanotic or dyspneic.* She had *a normal temperature on the* fourth day and it remained so except for a mild gastrointestinal upset on the fourteenth postoperative day with nausea, vomiting, and diarrhea. This responded promptly to the usual measures and intravenous fluid. The wound healed per primam. She was ambulatory on the twenty-first day and was discharged home on the twenty-eighth postoperative day. Table I shows the progress of the blood picture before and after surgery.

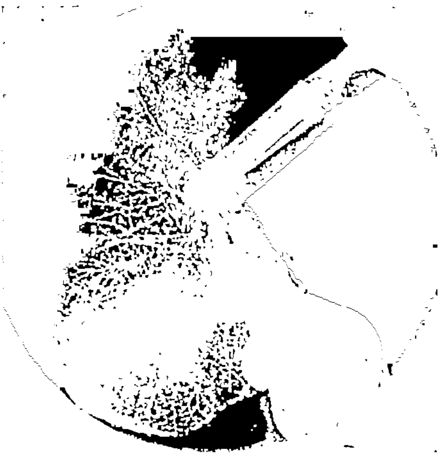

FIG. 5.—Roentgenogram of specimen (right lung) after injection of opaque solution into the inferior pulmonary vein. The aneurysmal sac filled promptly as did the branches of the pulmonary artery demonstrating the fistulous communication.

Pathologic Report.—The specimen (Figs. 5, 6, and 7) was the right lung which had been amputated just above the upper lobe bronchus. There were two lobes, an upper and lower, with a distinct fissure. There was no middle lobe. The lower lobe appeared normal. The inferior pulmonary vein was of normal caliber. The right upper lobe had many blue, thin-walled varicosities over its anterior surface and there appeared to be a very thin-walled sac filled with dark blood projecting from the lower aspect of the upper lobe at the site corresponding to where the middle lobe should be. This sac was 7 by 7 by 8 cm. and appeared to be an extension downward of the pulmonary artery. On the anterior inferior aspect of the sac there was a calcified plaque 2 by 2 cm. There were also two small calcific deposits in the lingula of the right upper lobe just above the sac. The pulmonary artery at its opening just above the upper lobe bronchus was 4 cm. in diameter. It was found to be widely dilated and it extended downward to the lower tip of the upper lobe, the diameter of this vessel being 3 cm. at its upper extremity and 2 cm. at its lower extremity and finally ending in a bulbous tip 3 cm. in diameter where there was an opening 8 mm. in diameter which extended downward terminating in the large sac previously described. When the large, globular sac was opened it was found to be full of unclotted blood and extended

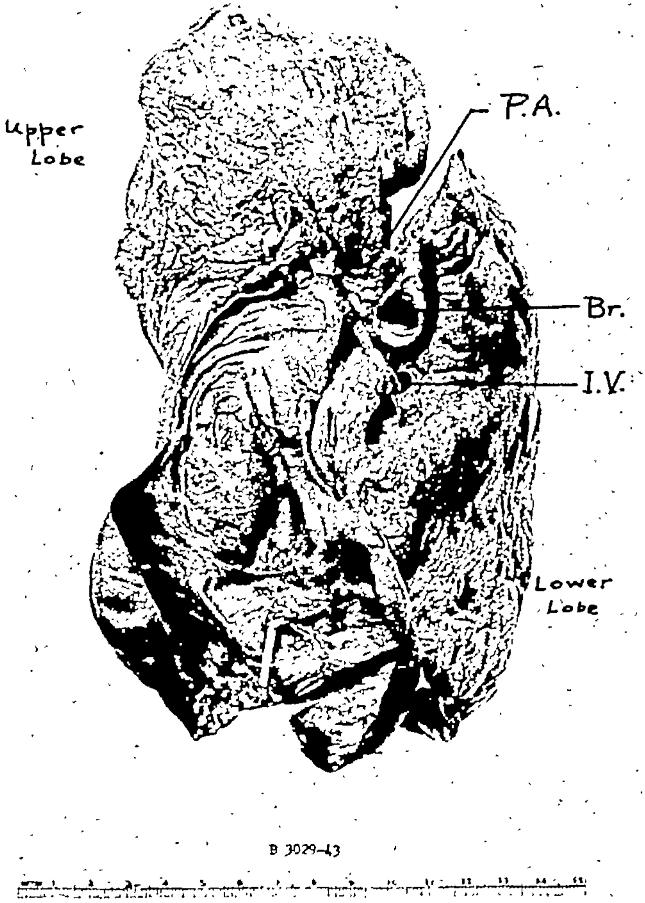

Fig. 6.—Photograph of mediastinal aspect of right lung. The pulmonary artery (*P.A.*) empties into a large, thin-walled sac which in turn communicates with another sac on the lateral aspect (see Fig. 7) through an 8 mm. fistulous opening through which an applicator stick has been passed. The inferior pulmonary vein (*I.V.*) is seen just below the main bronchus (*Br.*).

up in the lung parenchyma to a level of about the upper lobe bronchus and there ended blindly. In the larger, more anteromesially located sac which was seen to extend up in the lung parenchyma, there were multiple valvelike openings, some of which seemed to end in blind sacs; *others communicated with the superficial varicosities, and others* apparently communicated with the branches of the inferior pulmonary vein. No superior pulmonary vein was found in the specimen. The inferior pulmonary vein when injected with barium was found to communicate with the sac, for the barium passed back up through the branches of the vein and finally ended in the large aneurysmal sac.

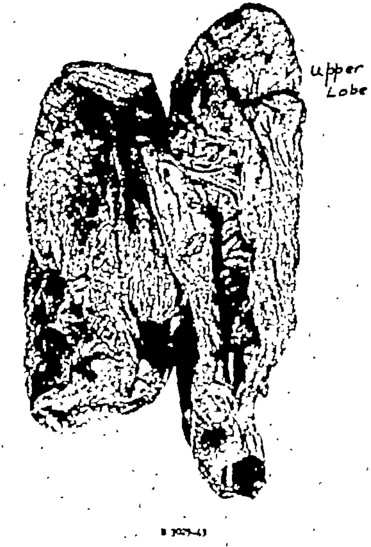

Fig. 7.—Photograph of lateral aspect of specimen showing stick passing from mesial inferior sac to another lateral sac in which are seen multiple small openings through which it presumably emptied itself.

One got the impression that in this specimen there was a large distended and dilated pulmonary artery which communicated at its inferior extremity with another, more anteriorly located, thin-walled aneurysmal sac which appeared to end blindly in about the mid-upper lobe but which drained through and emptied back into the lung parenchyma through multiple openings and eventually emptied into the inferior pulmonary vein. Diagnosis was arteriovenous fistula of the lung.

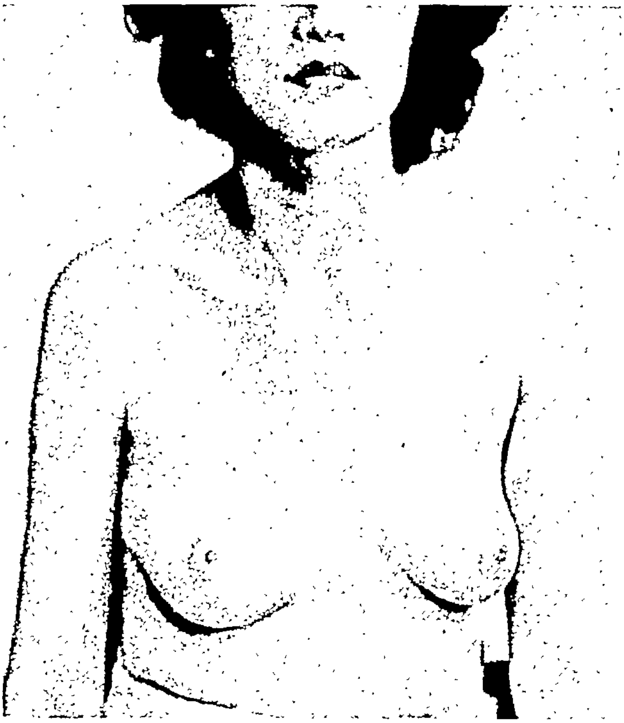

Fig. 8.—Photograph of patient four months after operation. The inframammary scar is concealed by the right breast and there is some retraction of the right thorax; otherwise there is no deformity.

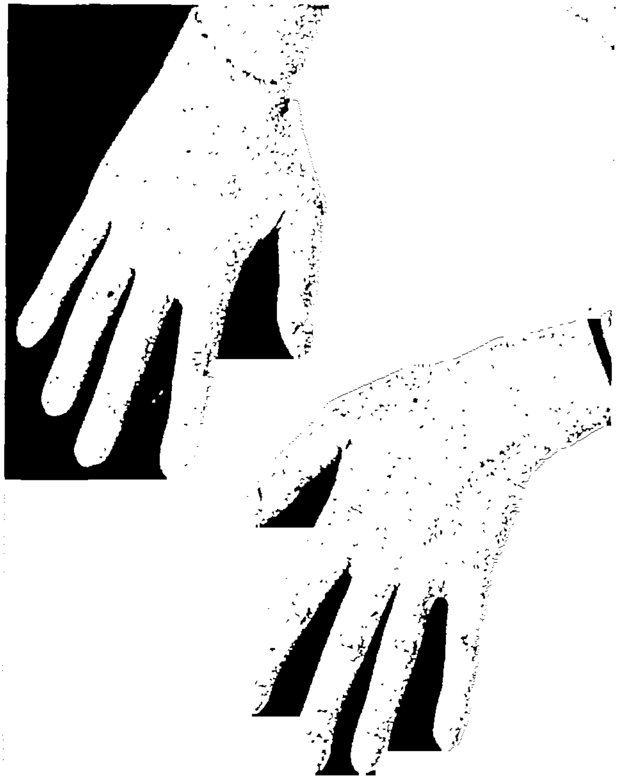

Fig. 9.—Photograph of hands four months after operation reveals the clubbing had receded about 50 per cent or more.

Jan. 10, 1944, almost four months after operation, the patient returned to the office for checkup. She reported that she was well, leading an active life, dancing, and was free of breathlessness. At the end of an active day sometimes she felt a "tiredness" in the right shoulder. Cyanosis had not been evident and she had noted a marked recession of the clubbing of the fingers and toes. She looked perfectly well. (See Fig. 8.) Cyanosis and dyspnea were absent. The conjunctivae were of normal color. There was no jugular distention. The heart sounds were normal and no murmurs were heard. The right upper anterior chest was depressed; the right chest showed no respiratory movement, was flat to percussion, and the breath sounds were absent. The blood pressure was 110/70. The liver and spleen were not felt. Edema was absent. Clubbing, while present, was definitely less in degree (Fig. 9).

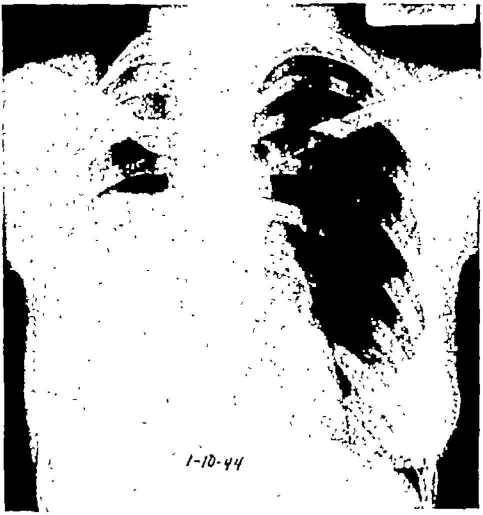

Fig. 10.—Roentgenogram of chest almost four months after operation showing thickened pleura with air above a fluid level at the first interspace.

Fluoroscopy showed a small, vertical vigorously pulsating heart, without conus fullness. The right chest was almost filled with fluid above which there was air, with level which was easily movable (Fig. 10).

In March, 1944, the patient was married.

DISCUSSION

As pointed out by Goldman[4] *all the reported cases* present clinical features and a symptom complex so similar that it is possible to describe a clinical syndrome which is unmistakably an arteriovenous shunt in the lung. Clubbing of the fingers and toes is always present. Polycythemia is a constant finding, its degree being in direct proportion to the size of the fistula. This is a secondary or symptomatic increase in red blood cells, with no increase in blood plasma, white blood cells, or platelets, and the blood volume may be increased to as much as 170 per cent, as in our case. It is distinguished also from true polycythemia by the absence of changes in the blood such as basophilic stippling and immaturity of the white blood cells. The oxygen unsaturation of the arterial blood is another constant finding. Goldman,[4] Hepburn and Dauphinee's[3] patients had an arterial oxygen saturation of 70 per cent. This test was not available in our case. The lack of any cardiac hypertrophy or pathology is due to the fact that the lesion is confined to the pulmonary circulation; this is an outstanding clinical feature. The presence over the tumor of a continuous murmur which is loudest at the end of systole and the beginning of diastole, and louder at the end of deep inspiration is of great aid in the diagnosis. Finally, of diagnostic importance is the finding of a chronic, nonprogressive pulmonary lesion which on ordinary roentgenograms appears as a solid mass, but which by planigrams is seen to be a branching mass and connected to the pulmonic arterial tree. The symptoms of headache, faintness, thickness of speech, ease of fatigue, and dyspnea on exertion, referred to by Goldman as common, were absent in our case.

Rationale for Surgery.—The question may reasonably be asked why surgery should be performed or advised in a young patient with arteriovenous fistula in the pulmonary circulation when the lesion has imposed no incapacitation as in our case. The fact that rupture of the fistula led to sudden death in Rodes' case has its counterpart in the extreme thinness of the sac in our case. Trauma to the chest would surely have been poorly withstood. Thromboses secondary to the polycythemia present a hazard. Bacterial infection of the fistula similar to that seen in such fistulas in the systemic circulation, although not reported to our knowledge, are probably possible.

Ideally, one would hope to be able to remove the fistula without disturbing the lung. Failing this, one would hope to sacrifice not more than a single lobe. In our case a total pneumonectomy was imperative because there was absence of the superior pulmonary vein and the upper lobe arteriovenous aneurysm was emptied through the inferior pulmonary vein. Experience with this case leads us to believe removal of the fistula without sacrificing at least one lobe is only occasionally to be accomplished.

SUMMARY

1. Arteriovenous fistula of lung produces a syndrome characterized by cyanosis, clubbing of fingers and toes, symptomatic polycythemia,

and symptoms of anoxemia, usually in a young patient with an obscure lung tumor and a normal heart. A continuous murmur may be heard over the tumor.

2. The treatment is resection of the fistula or pulmonary resection, either of the lobe or the entire lung.

3. The case of a patient with arteriovenous fistula of the right lung cured by pneumonectomy is reported in detail.

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DISCUSSION

DR. WILLIAM E. ADAMS, Chicago, Ill.—I enjoyed very much hearing this excellent presentation of Dr. Jones' case. The increasing number of reports of this condition speaks for its greater frequency than has been believed in the past.

In all probability a number of fistulas of the lung have gone undiagnosed and the patients have been treated for polycythemia vera. I would like to present the slides of a case which first came to our attention about one year ago.

(Slide 1.) The patient was a 24-year-old man who came for treatment of repeated attacks of epistaxis. He gave the history of having had clubbing of the fingers and toes and marked cyanosis for a period of about sixteen years or when he was about 8 years of age. He had worked on a farm until about one year before his admission and had not been really incapacitated by the lesion. He lost little weight, although he was relatively thin.

The other symptoms which he presented were few outside of having those changes which can be seen on his lips, hemangioma on both lips. The clubbing and cyanosis were as marked on the toes as on the fingers.

(Slide 2.) On examination, little could be found other than what was seen in the first picture. There was, as in Dr. Jones' case, no evidence of a thrill or murmur over the area in which the irregular opacity in the left lung mid-field can be seen. It is impossible to see also a very small opacity which was present in the right lung field. This picture is one taken on admission and is similar to the first which was made two years before admission. There were very marked changes in the lung and long bones of the arms and legs; marked sclerosis and periosteal new bone formation. This was evident even down in the metatarsus and phalanges of the feet.

(Slide 3.) On examination of the blood, as in Dr. Jones' case and all these cases, there was a marked increase in the hemoglobin and cellular content. You see here the blood volume is practically double, all of the increase being the cellular content. The hemoglobin was 23 Gm. and the red cell count 7,200,000.

(Slide 4.) The oxygen studies, as you notice, were very interesting. Although this man was markedly cyanotic he had 25.1 volumes per cent of oxygen in the arterial blood with a capacity of 35.5 volumes per cent. In other words, there was an enormous compensatory change. The diagnosis of the condition was made by my associate, Dr. Thornton, on the basis of the marked clubbing, cyanosis, the x-ray shadow, and no evidence of cardiac disease.

A left pneumonectomy was done, and at the time no attempt was made to replace the blood loss which was about 2,000 c.c. Instead, it was replaced by plasma for we didn't know how much ill effect there would be by the sudden reduction in the blood volume. I won't go into all the details, but following operation there was an immediate fall in red cell count and hemoglobin content, and a restoration of the oxygen values to approximately normal. The patient made an uneventful recovery and has been working since. This operation was made one year ago last February.

DR. ROBERT M. JANES, Toronto, Canada.—Actually we have had only three cases. The first case (operation by Dr. Shenstone) was similar in every way to the case that Dr. Jones has reported except that there were such great leashes of vessels about the hilum that a pneumonectomy by the tourniquet technique was necessary. A brief attempt at dissection made it obvious that if this method were followed the result would be a fatal hemorrhage. Fortunately, healing by primary union occurred and no bronchial fistula developed. The patient has since had two children.

The third patient had a single lesion in the middle lobe and was treated successfully by a dissection lobectomy. It is the second case that I thought might be interesting to you, particularly as the pages of the *British Journal of Surgery*, in which it was reported recently, may not be accessible to many of our members. This patient was a young man 30 years of age, who had been well except that he had always been somewhat lacking in energy. Two years before I saw him, while in the reserve Army, he ran across the camp and suddenly coughed up a considerable quantity of blood. This was repeated twice in the succeeding weeks and he was discharged. The x-ray picture was said to be normal. There were no further symptoms until three weeks before I saw him, when after moderate exertion he again had a very severe hemoptysis (slide 1). The roentgenograms at that time were as you see. There was a large lesion in the right lung and one small one. There were two small lesions in the left lung. This case was an exact duplicate of Rodes' even to the presence of multiple small submucous hemangiomas on the lips. There were no blood changes. Oxygen saturation studies were, unfortunately, not done because of wartime shortages of staff. The right side was operated upon first. Upon opening the chest the diagnosis became obvious. Since the lesions were bilateral the problem presented was different.

It was decided to attempt local resections. The large lesion was about the size of a hen's egg. The vessels entering it, each about 4 or 5 mm. in diameter, were individually isolated and ligated. It was then easy to remove the tumor without bleeding. The smaller one was removed by simply placing clamps on either side of it and excising the tissue between them. The defects in the lung were oversewn. The patient recovered satisfactorily. Six months later the left chest was opened and local resections of the two lesions in the lower lobe carried out. He again made a good recovery and has remained well.

I should like to point out that the polycythemic changes may occur late in these patients. In our first patient we know that these changes were not present only a little more than one year before she was operated upon even though her symptoms dated back to when she was 12 years of age. Neither of the other patients had polycythemia. Since there is danger from hemorrhage the development of this condition should not be awaited.

I would suggest that we keep hemangiomas in mind as a possible explanation of serious pulmonary bleeding not otherwise explicable. I have operated upon one other patient for recurring massive hemorrhage from an upper lobe that was certainly going to prove fatal. The pathologists failed to discover the source of bleeding. I am now convinced that they overlooked one of these lesions. I have recently seen a section of a hemangioma of a lung accidentally discovered in routine autopsy. Had the block for section not included it by mere chance, its presence would not have been suspected.

DR. I. A. BIGGER, Richmond, Va.—I would like to raise a question, but am not certain as to the advisability of the procedure I have in mind. It seems to me that in some of these cases ligation of the branch of the pulmonary artery to that lobe should be considered. I am, of course, fully aware of the inadvisability of ligation of the artery alone in arteriovenous fistulas involving peripheral vessels, but I believe this is a different problem. While there are communications between the bronchial arteries and the pulmonary veins, there are few collateral arterial channels, so proximal occlusion of the artery might cure the fistula.

DR. JOHN C. JONES, Los Angeles, Calif.—In answer to Dr. Bigger's question, this was a hugely dilated, thin pulmonary artery, and after we passed a tape around it, it seemed as though the tumor mass did not diminish in size after the tape was drawn taut in order to close off the blood supply passing through the pulmonary artery. I did not feel justified in trying to dissect out the branches of the pulmonary artery because the branches seemed almost as large or larger than the normal size of the average pulmonary artery. The fact that there was no superior pulmonary vein to be found and that the inferior pulmonary vein appeared to carry all the blood back to the left heart was even more important. These two considerations led me to believe that it would be better not to attempt local resection and perhaps get a hemorrhage which could be controlled easily, I grant you, because I had a tape around the artery at its origin. I feel satisfied in my own mind that I would rather sacrifice the lung than attempt to dissect out any of the branches or even to leave the lung in and ligate the pulmonary artery.

INDICATIONS FOR PERICARDIOTOMY WITH SPECIAL REFERENCE TO THE EXPOSURE OF AN INFECTED PATENT DUCTUS

Harold Neuhof, M.D.
New York, N. Y.

INJURIES of the heart and hemopericardium, suppurative and constrictive pericarditis, foreign bodies, and neoplasms comprise the roster of conditions for which pericardiotomy with or without drainage may be indicated. They can be termed the standard indications for the operation. It can be assumed that pericardiotomy has been employed for a variety of other purposes which are not referred to in the literature. Since pericardiotomy is to be regarded as safe and free from untoward sequelae the possible usefulness of the operation for conditions other than the standard ones is worthy of consideration. I shall describe such conditions encountered in my own experience and hope that others will join in broadening the indications for the procedure. In all the conditions which will be described, with the exception of the patent ductus, pericardiotomy was at least begun as an exploratory procedure. The description of each of the conditions will be accompanied by an illustrative case report.

1. *Exploration Within Pericardial Sac of Hilar-mediastinal Lymph Nodes.*—In a number of instances of carcinoma of the lung there was considerable doubt at exploratory thoracotomy as to the extent of hilar node involvement. This was particularly the case when the lung was broadly adherent posteriorly or when the tumor in the lung abutted on the posterior mediastinum. In either instance considerable dissection would be required to ascertain the presence and extent of lymph node involvement; this would be unnecessary if the lesion were inoperable. Pericardiotomy and exploration within the sac supplied the required information.

CASE REPORT

H. N., a man 60 years old, not a good operative risk, had a history chiefly of pulmonary infection and positive biopsy from the bronchus of the left lower lobe. Exploration was done through a liberal transthoracic incision. Both lobes, especially the lower, were broadly adherent posteriorly. There was a substantial mass in the superior portion of the lower lobe which appeared to be partly neoplasm and partly inflammatory and was adherent to the parietes behind the hilus. Inspection in front of the hilus revealed several small lymph nodes which

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Read at the Twenty-fifth Annual Meeting of the American Association for Thoracic Surgery, Chicago, Ill., May 5 and 6, 1944.

were removed for biopsy. One was positive. Informative palpation of the posterior aspect of the hilus was not possible. The pericardium was incised freely and palpation within it revealed a series of obviously involved nodes at the hilus and in the mediastinum, rendering the lesion inoperable. The pericardium was not sutured, but would have been sutured if the lesion had been operable. The thoracic wound was closed. The postoperative course was uneventful.

2. *Decompression of Serous Pericardial Effusion. Exploration.*—Upon a number of occasions obvious accumulations of fluid within the pericardial sac are to be noted at thoracic operations performed for a variety of conditions. Whether or not they play any part in the presenting clinical picture usually does not come into question. However, in the following case pericardiotomy was performed because the accumulation of fluid apparently produced some measure of cardiac tamponade.

CASE REPORT

J. M., Jr., 24 years old, suffered from thoracic pain in the left side and unproductive cough for a year. Because of a clear left pleural effusion he was said to have been discharged from Military Service with the diagnosis of tuberculosis. A recent complaint was dyspnea and asthenia of three weeks' duration. Bronchoscopy revealed a stenosing lesion of the left upper lobe; biopsy suggested a malignant tumor. An exploratory left thoracotomy was performed. Soon after the chest was opened the blood pressure began to fall, the pulse to become small and rapid. An infiltrating tumor of the upper lobe intimately attached to or invading pericardium was noted. There were no involved anterior hilar nodes. The pericardium was tense and cardiac pulsations could scarcely be discerned. The pericardium was freely incised adjacent to the tumor and there was the escape under tension of a very considerable quantity of clear fluid. Immediately thereafter the pulse volume improved, the pulse rate slowed, and the blood pressure rose. Before incision of the pericardium it appeared doubtful if the patient's condition would permit of further investigation, to say nothing of eradication of the neoplasm. After drainage, the general condition was good enough to proceed, thus indicating that the clinical picture probably was due to a measure of cardiac tamponade which had been present for several weeks before operation and was presumably exaggerated by anesthesia. The further investigation in this case consisted of exploration through the widely opened pericardium. Posteriorly, neoplastic tissue (from which a specimen was removed and reported as anaplastic medullary carcinoma) was felt and visualized. It not only invaded the pericardium in this region but also appeared to invade the heart directly. The case was thus inoperable, the pericardium was left open, and the thoracic wound was closed. The postoperative course was uneventful and there was no return of the picture suggestive of cardiac tamponade. He left the hospital about two weeks after operation and died several months later.

3. *Exploration to Determinate Extent of Invasion of Pericardium by Pulmonary Neoplasm. Partial Excision of Pericardium.*—A report has been made before the American Association for Thoracic Surgery concerning a peripherally invasive type of pulmonary cancer which also is usually characterized by limited and late (if any) regional lymph node involvement. Metastases are prone to occur via the blood stream. Thus, the local situation is favorable for eradication in such cases provided it is possible to excise beyond the region (such as diaphragm and thoracic wall) of peripheral invasion. In the following case the lesion appeared at first to be inoperable because of fixation at the mediastinum and invasion of the pericardium. Exploratory pericardiotomy revealed the extent of the invasion. Liberal sacrifice of the pericardium made possible an adequate excision of the tumor.

CASE REPORT

M. F. B., a man 50 years old, was admitted with a history of asthenia, weight loss, cough of six weeks' duration, and pain in the left upper chest of two weeks' duration. On percussion there was dullness behind the sternum extending to the left side. In the roentgenogram there was seen a large mass apparently in the upper lobe and attached anteriorly. Bronchoscopy was negative. The operation was performed June 30, 1942. Extending from the anterior portion of the upper lobe was a tumor which occupied the anterior mediastinum and was tightly wedged in place. Adhesions were separated but the tumor remained immobile. In order to attempt to ascertain the possibility of removal and the extent, if any, of invasion of the pericardium, an incision was made through the parietal pericardium above the tumor. The tumor was found to invade broadly the parietal pericardium, bulging into the sac and compressing the heart. However, there was no invasion beyond the parietal pericardium. The latter was incised around the tumor sacrificing a section about three inches in each diameter. The tumor was thereby released and it was removed together with about one-half of the upper lobe by the tourniquet technique. (Microscopic report: squamous-cell carcinoma.) In order to cover the large pericardial defect the pulmonary tissue adjacent to the stump was approximated to mediastinal tissues medial to and above the pericardial defect.

It now is almost two years since operation. The patient is in good condition, symptom-free, and pursues an active occupation.

4. *Adhesive Pericarditis With Slight External Evidence. Exploratory Pericardiotomy Followed by Partial Pericardiectomy.*—In addition to the classical forms of constrictive pericarditis there are cases with similar clinical manifestations in which the constricting factor is not evident. To these I have attached the term "restrictive peri-

carditis." The point to be made here is that these may not be identified at operation by an inspection of the external surface of the pericardium. An exploratory incision of the pericardium is indicated. It can be added that the diagnosis of constrictive pericarditis is not always easy and that a symptom complex which appears to be more or less characteristic may be due to lesions quite unrelated to pericardial disease. Thus, there was an instance in which I explored the pericardial sac and found it to be free from abnormality. Subsequently the case came under the observation of White in Boston. The clinical picture warranted re-exploration of the pericardial sac by Churchill, again with negative findings.

CASE REPORT

N. B., a 23-year-old man, gave a history of abdominal distention for three months. Six liters of fluid were removed before admission. The abdomen was tense with fluid. The blood pressure was 126/96, the venous pressure 19 cm. After the customary preparation, operation was performed, July 19, 1940. The approach was a left transpleural intercostal one. The external pericardium was apparently not thickened although somewhat opaque. Cardiac pulsations were normal. The pericardium when incised was found to be very slightly thickened. The lesion was an adhesive fibrosing one of the visceral and not of the parietal pericardium. After the visceral membrane was generously removed the cardiac action was free but the heart did not bulge through the gap in the parietal pericardium.

The postoperative course was uneventful. There has never been a return of ascites. It is now almost four years since operation. The patient has remained symptom-free in a physically active occupation.

5. *Exploratory Pericardiotomy and Secondary Pericardiostomy for Suppurative Pericarditis.*—The suture of the margins of the opened pericardium to the thoracic incision facilitates greatly the drainage of an infected sac but does not always solve the problem. Thus, undrained collections may be found at autopsy. With this in mind the following patient was subjected to exploratory pericardiotomy after drainage for suppurative pericarditis had already been instituted.

CASE REPORT

B. S., a boy 5 years old, had been operated upon at another institution for suppurative pericarditis as a metastatic focus derived from the humerus. At the operation several weeks before admission, when the patient's condition was desperate, a large quantity of pus under tension was evacuated. The general condition rapidly improved, the patient became afebrile, but rather profuse discharge of pus from the wound continued. At operation at Mount Sinai Hospital the pericardium was widely exposed extrapleurally and carefully incised without preliminary aspiration. Purulent exudate was encountered. Further dissection revealed not only additional purulent exudate but also surprisingly large collections of pus both in front and behind the heart. Painstaking irrigations were required over a period of weeks before the pericardial sac was clean and could be permitted to heal. It has remained healed.

6. *Pericardiotomy for Acute Pericarditis of Low-Grade Virulence.*—The following is the only instance in my experience in which an exploration disclosed an inflammatory lesion of obviously low-grade virulence.

CASE REPORT

C. K., a 68-year-old woman, was operated upon for diaphragmatic hernia at another institution, in October, 1942. A transthoracic operation was performed. It was followed by fever. Several thoracenteses were performed. Fever continued unabated. On admission in January, 1943, it was thought (roentgenograms difficult to interpret) that an intrapleural suppurative focus existed in the region of the pericardium. At operation, January19, a suppurative focus was not found. The surface of the pericardium appeared congested and possibly thickened. Aspiration revealed turbid yellow fluid (culture was reported later as sterile). The pericardial sac was laid open freely and found to be occupied by fibrinous exudate and small pools of yellowish fluid. The margins of the pericardium were sutured to the soft parts of the thoracic incision. The skin was strapped. (Report on specimen from pericardium: acute inflammation. No organisms found in sections.) Postoperative dressings consisted of unstrapping of skin wound, evacuation of clear fluid, and restrapping of wounds upon several occasions until healing occurred.

After a fever-free interval fever returned and another exploration for a suspected intrathoracic abscess was performed on February 19. None was encountered. The pericardial sac was opened and found to be free from infection. The patient was discharged in a fever-free interval.

7. *Pericardiotomy for Better Exposure of the Patent Ductus in the Presence of Subacute Endarteritis.*—The brilliant results achieved by Touroff and subsequently by others with the ligation of the ductus in the presence of subacute endarteritis were offset in a measure by occasional disastrous hemorrhage at the time of operation. The source of hemorrhage was the posterior surface of the ductus and the cause (in addition to the possible or probable friability of the ductus when the seat of endarteritis) was the difficulty of dissection in this region. It was apparent that although the ductus is outside, but immediately adjacent to, the pericardium the exposure of the structure would be facilitated by incising the pericardium and retracting the dilated pulmonary artery. This was first tried in a patient without endarteritis and, found to be useful for the purpose, was applied in two with en-

darteritis. One case is described. In view of current reports it should be stated that a course of penicillin must be tried before subjecting patients with patent ductus to ligation during the period of subacute bacterial endarteritis.

CASE REPORT

M. N., a woman 30 years old, was admitted Sept. 27, 1943, with a three months' history of fatigue and fever. The diagnosis of subacute bacterial endarteritis was established elsewhere and full dosage of sulfadiazine was administered. There had been one episode of pulmonary infarction. On admission there were noted the characteristic machinery murmur and phonocardiogram. The blood culture was positive for *Streptococcus viridans.* At operation the mediastinal pleura was incised in the customary manner, and the recurrent nerve visualized beneath the aorta. The ductus was found to occupy a lower position than usual and was short and extremely wide (1½ cm. long, 2 cm. wide). The dissection for its release was begun on the superior and inferior surfaces but it soon became evident that dissection under vision would not be possible on its deep surface. In the hope of obviating more or less blind and dangerous dissection in this region mobilization of the enormously dilated pulmonary artery was decided upon. This was made possible by incising the pericardium radially and freely over the region of the left auricular appendage. The incision was prolonged to the beginning of the ductus. A gauze pad was placed over the pulmonary artery which was drawn downward and medially. The deep surface of the ductus was visualized thereby and its dissection was readily accomplished. Two silk ligatures closed the ductus with disappearance of the thrill. The pericardium was not sutured. Blood pressure before ligation was 86/64, after ligation 100/70.

There was an accumulation of bloody pleural fluid after operation of which 350 c.c. were aspirated. There were also two chills in the first postoperative days. Otherwise, convalescence was satisfactory and all blood cultures were negative, including the one immediately after ligation of the ductus. It is now seven months since operation. The patient is symptom-free and does the active work of a housewife.

DISCUSSION

DR. STUART W. HARRINGTON, Rochester, Minn.—I have not used pericardiotomy in the treatment of ductus arteriosus, but for the past two years I have used a posterolateral approach for exposure in the surgical treatment of ductus arteriosus both in the infected and noninfected ones.

I believe that this approach has very definite advantages over an anterior approach. The exposure is more adequate. It is particularly advantageous if the pleura is adherent since the lung can be separated from the apex permitting exposure of the ductus. It is also very adequate in the infected patent ductus where there are many adhesions and enlarged lymph nodes around the ductus. It has the advantage of a bilateral approach to the ductus. If hemorrhage should inadvertently occur during the separation of the ductus, the surgeon is in a position to control it by compression of the fingers until the ductus can be ligated, which I have found necessary to do in one of my cases.

The posterolateral incision is made around the angle of the scapula with resection of the posterior two-thirds of the fourth rib and in some instances an additional exposure can be obtained by resecting a small segment from the angle of the adjacent ribs. The pleural cavity is entered through a posterior periostomy of the fourth rib.

DR. JOHN C. JONES, Los Angeles, Calif.—I have not opened the pericardium of any of the patients in whom I have ligated the patent ductus arteriosus. Gross of Boston, who has had the most experience with the ligation of the ductus arteriosus, makes a very definite point, and I think a very good one. I feel certain he was referring to the ligation of the ductus arteriosus in patients with infection. He believes there is a definite disadvantage in opening the pericardium because there is a trickle of pericardial fluid through that opening which obscures the field of the dissection, and I have not seen any noninfected ductus arteriosus which required the opening of the pericardium for ligation.

I should like to make one point, however, that the more ductus one ties, the greater variety of dissections one encounters. I think in some cases the ductus appears short because of the reflection of the pericardium up over the ductus, extending to the aorta. Some that are not infected, where there are no enlarged lymph glands, appear as though there had been infection, even in young children. Going anteriorly and antromesially toward the right main stem bronchus, the dissection is difficult. If one carefully dissects down, removing and cutting these fibrous reflections from the pericardium up toward the aorta, one is surprised how much longer the ductus arteriosus actually is than it appeared originally when the dissection was started.

I quite agree with Dr. Gross that there is a definite disadvantage to having the pericardium opened in the region of the ductus because of the trickle of fluid. There is another point I would like to make as far as opening the pericardium is concerned in malignant tumors.

About one and one-half years ago I operated upon a boy about 9 years old who had a recurrence of an upper abdominal wall tumor which had been reported as a fibrosarcoma and which obviously not only involved the abdominal wall, but also the costal cartilages. After removing the large abdominal wall tumor and dissecting upward to resect the costal arch on the left, it was seen that the tumor involved the left diaphragm anteriorly and the left pleural cavity, so that was removed. Then the dissection was made mesially and posteriorly, and it was found that the pericardium was involved. Therefore, I had to remove the lower two-thirds of the anterior pericardium.

Then, extending over to the right, it was observed that the anterior right leaf of the diaphragm and the right pleura anteriorly were infiltrated with new growth, and had to be removed. The dissection was carried on downward so that I had all four cavities opened widely at one time. It was a beautiful demonstration of controlled anesthesia. It was necessary to use the liver in order to close the wound, but the liver makes an admirable closure of the abdominal wound, provided there has been enough fibrosis from the previous operation in order to stitch it to the remains of the abdominal wall anteriorly. This patient had an uneventful convalescence and is well thus far.

DR. CAMERON HAIGHT, Ann Arbor, Mich.—The indication for partial resection of the pericardium in conjunction with the removal of tumors of the lung is rare. The first instance that I am aware of is one mentioned by Clarence Crafoord of Stockholm in his monograph *On the Technique of Pneumonectomy in Man*, 1938.

I would like to call your attention to an interesting observation in a patient for whom a pneumonectomy with the resection of a large area of the pericardium was performed three years ago. The lesion, which proved to be a squamous-cell carcinoma, was situated in the lingular portion of the left upper lobe. The diaphragm was moving two weeks before operation, but on the day before operation, there was complete diaphragmatic paralysis. As the involvement of the phrenic nerve was recent, operation was done in spite of the fact that local extension of the tumor to the pericardium and phrenic nerve was anticipated. It was hoped that the extension was sufficiently recent, so that it would be possible to remove the local metastases in conjunction with the lung. An anterior approach was used, and the tumor in the upper lobe was found to be adherent to and invading the pericardium over an area about 4 by 5 cm. in diameter. The phrenic nerve was involved in the growth.

The pericardium was opened and an encircling incision about 1 cm. wide was made of the neoplastic infiltration in the wall of the pericardium. The involved area of pericardium was left attached to the lung and was removed in conjunction with the lung. After fixation in 10 per cent formalin, the resected area of pericardium measured 6.4 by 5.4 cm.

During the removal of the lung, the exposure of the pulmonary pedicle was maintained by the assistant's hand which incidentally maintained slight pressure against the heart, thus keeping it in place. On releasing this pressure after removal of the lung, the heart immediately herniated through the large opening in the pericardium, *and the apex of the heart almost touched the thoracic wall laterally*. To describe the phenomenon more vividly, the heart actually flopped out of the pericardial cavity. This resulted in a sudden kinking and torsion of the vessels at the base of the heart. The blood pressure became inaudible and the pulse imperceptible. By pressing upon the heart it could be returned into the pericardial cavity and by tilting the patient to the contralateral side at an angle of about 20 degrees, the heart remained within the pericardium. As soon as the heart was returned to its normal position, the blood pressure and pulse rate returned to the levels that were present before the incident occurred. The opening in the pericardium was much too large to be closed by suture. Although the use of a free fascial graft was considered, this possibility was abandoned because of the additional time that would be required and the unforeseen difficulties that might be encountered. More importantly, it was believed that the herniation of the heart could be prevented by keeping the patient turned on or toward the contralateral side.

After closure of the wound, a loud clicking noise, similar to a knock, could be heard and felt with each heartbeat. During the first day after the operation the intensity of the sound was so great that it could be heard when one was standing in the doorway of the patient's room, the patient being at the opposite end of the room at a distance of about twelve feet. The intensity of the sound gradually subsided during the next several days and then completely disappeared coincident with organization of the pleural fluid within the empty pleural cavity.

It was necessary to keep the patient inclined toward the contralateral side after operation. At one time during the first few hours after operation, the patient was inadvertently allowed to be flat on his back, and he immediately developed severe shock, which was promptly corrected by again turning him toward the right side.

As I do not recall that this phenomenon of herniation of the heart, in a patient for whom a large pericardial opening was not closed, has been described previously, I felt that it would be of interest to mention it as well as the use of posture in controlling this complication. Although three years have elapsed since operation, the patient is well and he has no evidence of cardiac embarrassment or recurrence of the tumor.

DR. EVARTS A. GRAHAM, St. Louis, Mo.—A number of years ago Sauerbruch described the procedure of leaving the pericardium open for drainage into the pleural cavity as in cases of intractable pericardial effusion. I had occasion about four years ago to carry out that procedure in a woman who had a recurrence of a carcinoma of the breast in the pericardium with massive bloody effusion in the pericardium which required frequent tapping. By the simple procedure of removing some of the pericardium and letting the rest of it drain into the left pleural cavity the woman was perfectly comfortable for a period of about two years before she died, finally, of further recurrences.

This was an exceedingly simple procedure and one which gave great comfort to the patient.

DR. JEROME R. HEAD, Chicago, Ill.—I had one rather peculiar accident incident to opening and closing the pericardium. I believe that it might be worth while to report it. The left auricular appendage extends down beside the arterial cone. At each heartbeat this thrusts forward.

In taking out a pericardial cyst, I tore the pericardium, and at the end of the operation, which was a perfectly simple one, I started to close it with a running stitch. Through the early part of the closure I could see the needle but was unable to watch it during the last stitch. This was followed by a tremendous gush of blood through the remaining opening and into the pericardium. I opened the sac widely and found that my needle had torn a piece out of the auricle which had come down and hooked my needle. It emitted a stream of blood about the size of a lead pencil.

I put my finger on it and inserted a stitch in the end of the flap for traction. The stitch immediately pulled out. Fresh bleeding developed at the site of the stitch. I then tried to grasp it on the side with a hemostat and ended by having more bleeding. My assistant finally suggested that I take an ovum forceps and grasp the whole appendage, which I did, and then ligated back of the forceps. This controlled the bleeding. The patient made an uneventful recovery except for two or three weeks when he had almost daily attacks of paroxysmal tachycardia. I believe that this is worth reporting as being the only way in which one can control bleeding from this part of the auricle.

DR. FRANK S. DOLLEY, Los Angeles, Calif.—There is a point that has not been emphasized despite the splendid discussions of this paper. Following pericardiotomy there may be no apparent counterindication to closure of the pericardium. It should be borne in mind, however, if there is any trauma at all, serum in considerable amount may collect in the pericardial sac. If the pericardium is completely closed, tamponade may occur and, under the care of an intern unused to this complication, lead to the patient's death.

Even if the pericardium is left open, unless there is some provision for drainage other than leaving it open, an accumulation of serum in the immediate vicinity of the opening of the pericardium may occur leading to a backup into the pericardial sac and to serious or fatal tamponade. In other words, some type of drainage (rubber tissue) to the outside or into the pleural cavity in cases where the serum is likely to form in considerable quantity is a necessary safeguard.

A tamponade developing during the night might lead to death without the nurse being aware of change for the worse.

THE MANAGEMENT OF STRICTURE OF THE ESOPHAGUS

WITH SPECIAL REFERENCE TO COMPLETE STRICTURE OF THE MID-ESOPHAGUS WITH A DETAILED REPORT OF TWO CASES

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THE common conditions of the esophageal wall with obstruction are cancer of the esophagus, esophageal diverticulum, cardiospasm, web (both with and without the Plummer-Vinson syndrome), cicatricial stricture secondary to caustic burns, cicatricial stricture secondary to peptic ulcer or esophagitis often associated with peptic ulcer below the esophagus, cicatricial stricture secondary to or associated with surgical procedures, cicatricial stricture secondary to tuberculous glands of the mediastinum, and congenital developmental anomalies, named in the order of greatest frequency.

The method of treating esophageal strictures is dilatation of the narrowed area. A satisfactory result is obtained when the dilatation is sufficient to allow the passage of a regular diet without difficulty. Until this is accomplished, the diet may be altered to admit its passage through the narrowed esophagus.

The amount of dilatation or distention that may be obtained in tissues by the application of pressure is, to say the least, amazing. In the esophagus we see diverticula that have become as large as a grapefruit, and the esophagus may attain a circumference of ten inches as the result of continued pressure from its fluid content and by the added pressure exerted by the muscles of deglutition.

The passage of any instrument through the esophagus is fraught with danger for trauma to the esophageal wall may result in mediastinitis, while perforation or rupture produces mediastinitis except in rare instances. The mediastinitis spreads rapidly and terminates fatally in almost all instances unless radical measures are taken to drain the mediastinal infection and limit its spread. In such cases the judicious use of the sulfonamides and penicillin must not be overlooked.

The frequency and the amount of dilatation vary with the individual case, and this is governed by the experience and judgment of the attending physician. Once dilatations are begun, the tragedy of breaking of the esophageal wall reflects upon the judgment of the operator.

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CARCINOMA

Since dilatation can in no way be a cure for carcinoma, it may be used in giving palliative relief, improving nutrition and delaying gastrostomy or death. Dilatations have been carried out both with the esophagoscope and with the previously swallowed thread as a guide, obtaining pronounced relief over a period of time.

PHARYNGO-ESOPHAGEAL DIVERTICULUM

The only logical treatment of these diverticula is surgical removal. Symptoms of a small pharyngo-esophageal diverticulum may be relieved to some extent by dilatation of the upper end of the esophagus.

We believe that dilatation of the upper end of the esophagus following operation decreases the tendency toward recurrence of esophageal diverticula, and in a few cases dilatations of this area have been necessary due to narrowing following the operative procedure.

CARDIOSPASM

A review of the etiologic theories of esophageal achalasia is unnecessary. There is narrowing or stenosis of the lower end of the esophagus which produces symptoms that may be relieved by dilating the narrowed area. In 112 cases of cardiospasm requiring treatment, we encountered 4 with advanced torsion, in which dilatation failed to give satisfactory relief, and operative treatment was advised. Yet 3 of these 4 patients have managed to exist with considerable discomfort, and 1 died of pneumonia several weeks after a gastrostomy had been performed to maintain nutrition. The remainder were dilated with control of most of their symptoms.

For years it was our practice to visualize the lower end of the esophagus through the esophagoscope, balloon the esophagus and pass dilators, and usually the esophagoscope, into the stomach. At this time a Tucker-Plummer bag was passed through the esophagoscope, the esophagoscope was withdrawn, and the Tucker bag was dilated to eight pounds pressure or until severe pain was produced. Following the esophagoscopic procedure, a modified Plummer bag, measuring one inch in diameter, was placed in the narrowed area under fluoroscopic guidance. It was inflated until pain was produced or until eight pounds of pressure was reached. The dilatations were carried out at intervals of one to three weeks until considerable improvement in the symptoms had occurred. If the symptoms were not relieved, the pressure was increased gradually over a period of months until twelve or even fifteen pounds of pressure was used. Following the suggestion of others, we began applying the higher pressure early in the course of treatment, and found that the results were much better in that the time required and the number of treatments were greatly decreased.

This year suppurative mediastinitis occurred in 2 cases following such dilatation. These are included in the 4 cases of mediastinitis to be mentioned later in this communication. Although both patients recovered after prompt surgical drainage, we were convinced that such rapid dilatation, while effective, was too dangerous for further use. Henceforth, we shall follow our former policy of gradual dilatation, accepting the inconveniences of increased frequency of dilatation and the time required rather than risk esophageal rupture.

Leakage occurred once under general anesthesia, which was given at the insistence of an extremely nervous and temperamental patient. In addition, too great pressure was used. This complication must be classified as secondary to an error in judgment, as the pain reflex should be preserved as a safeguard during esophageal dilatation for cardiospasm.

WEB

Webs with and without the presence of the Plummer-Vinson syndrome have been encountered in 42 cases. The treatment of web is usually dilatation; however, a few have been removed with a punch forceps. In many cases the initial dilatation relieves the patient indefinitely, although a few require repeated treatment. One patient returned six years after dilatation with further difficulty in swallowing. She was found to have a carcinoma in the upper esophagus.

CHEMICAL BURN

Sixteen patients with stricture of the esophagus resulting from chemical burn have been treated with relief of symptoms, 2 of which are reported here in detail. Four other patients have been seen, but went elsewhere for treatment or rely on a gastrostomy for feeding.

None of these patients was seen early or during the acute stage of the burn. They came to us weeks, months, or even years later, when discouraged by the lack of progress, or in difficulty either from their own neglect of treatment or inadequate treatment.

The only really troublesome difficulty encountered was re-establishment of an opening in a completely stenotic esophagus. If the patient was able to swallow liquids, no trouble was encountered. Once a guiding thread was passed through the strictured area from a gastrostomy opening through the mouth, persistent gradual dilatation accomplished a satisfactory result. The Tucker retrograde rubber bougie was used in the early phase of treatment of the most difficult cases.

PEPTIC ULCER AND ESOPHAGITIS

Strictures associated with peptic ulcer of the lower end of the esophagus or a severe esophagitis associated with peptic ulcer below the esophagus have required repeated dilatations in 15 cases. One patient was completely *obstructed and had been maintained on parenteral* feedings for three weeks before coming for treatment. A gastrostomy was done and retrograde dilatation carried out. In the remaining cases, after appropriate esophagoscopy, biopsy, and diagnostic studies, the swallowed thread was used as a guide for the repeated dilatations. All of these patients were greatly improved for long periods, some being entirely symptom-free. Two had a return of symptoms after being well for five and seven years, respectively. It is interesting to note that a large percentage of these patients with peptic ulcer of the lower end of the esophagus had a *short esophagus and a small diaphragmatic hernia.*

SURGICAL PROCEDURES

As a sequel to the treatment of carcinoma of the stomach by total gastrectomy and esophagojejunostomy in 68 patients, 6 patients developed a partial stricture at the anastomosis within a few weeks or months after the operation. All responded to repeated dilatations and here again a swallowed thread was useful as a guide. We have seen 2 patients in whom a portion of the cervical esophagus, 5 cm. and 1.5 cm., respectively, was removed accidentally during thyroid surgery. Complete obstruction had existed for six weeks in 1 case and for one year in the other.

Fortunately, neither patient developed a spreading mediastinitis, which may well be explained by the open drainage of the thyroid wound. Treatment was carried out by an open operation of the area of the cervical esophagus with peroral and retrograde esophagoscopy. In the patient with the smaller defect, a flap of mucous membrane was turned down from the upper segment and sutured in continuity with the mucous membrane of the lower segment. A thread was placed for retrograde dilatation.

In the patient with the larger defect, a thread was placed without any attempt to bridge the defect with mucosa. Retrograde dilatations *have been carried out and, surprisingly enough, this patient is able to* eat a normal diet; but dilatations to a No. 41 French bougie are still being carried out at three-week intervals over a previously swallowed thread.

In a case worthy of mention an early carcinoma was diagnosed in the upper end of the esophagus. An open operation through the neck was carried out, removing the entire carcinoma, and leaving a strip of mucous membrane approximately 6 mm. by 5 cm. connecting the hypopharynx and the esophagus. Repeated dilatations were carried out following this procedure, and at this writing, eleven years later, the patient is asymptomatic, and a 7 mm. esophagoscope can be passed with ease through the upper esophagus.

TUBERCULOSIS OF THE MEDIASTINAL GLANDS

Tuberculosis of the mediastinal glands is not uncommon and often produces a traction diverticulum. In a relatively few cases, however, a scar tissue stricture is produced which requires repeated dilatations. Five such patients have been treated, 1 of whom required dilatations at intervals over a period of seven years before she became asymptomatic.

CONGENITAL NARROWING OF THE ESOPHAGUS

In 2 cases a congenitally narrow esophagus was present. One patient was a girl 9 years of age, and the other was a man of 49 years. Both had lived their entire lives on fluids and strained foods, never having taken a normal meal. Both responded readily to repeated dilatations.

REPORT OF CASES

CASE 1.—*Nov. 24, 1942, a 60-year-old man accidentally swallowed a* quantity of wood-staining compound. He recovered promptly from the severe systemic shock, but during the next two months he developed a complete esophageal stricture. He lost twenty pounds in weight. For several days prior to admission all intake by mouth was regurgitated and he was sustained by intravenous fluids.

He entered the hospital Feb. 23, 1943, three months later. The general physical examination was not remarkable, except for dental caries and gingivitis. Gastrostomy was advised but refused. February 25, esophagoscopy demonstrated complete obstruction at the middle third of the esophagus with some ulceration of the wall above the stricture. The lumen was represented by a dimple of granulation tissue, through which a No. 10 Jackson bougie was passed without difficulty. Larger dilators, up to No. 16, were then passed. Following dilatation the esophagus was easily inflated with air, and a swallow of warm water seemed to go through without apparent discomfort to the patient. A few minutes later a sharp lancinating pain in the upper abdomen occurred. Because of the possibility of perforation and emphysema of the mediastinum, roentgenograms were taken which showed no sign of mediastinal disease.

Steady boring pain under the right costal margin and xiphoid process persisted, but the temperature and pulse rate, after initial elevation within the first eight hours, subsided to normal in eighteen hours, and serial white blood counts at two, six, and eighteen hours were 6,900, 19,900, and 17,000, respectively. At thirty hours the temperature was 101° F., pulse rate 120, and the leucocytes numbered 31,000. Roentgenologic examination with a portable machine showed an elevated right diaphragm. The pain had increased and was made worse by deep inspiration. The diagnosis of acute suppurative mediastinitis was obvious.

February 26, thirty-four hours after esophagoscopy, a right posterior mediastinotomy through the bed of the eighth rib yielded 600 c.c. of thin foul-smelling pus containing anaerobic hemolytic streptococci.

The patient recovered after a prolonged convalescence. A gastrostomy was established on March 5, eight days after the mediastinotomy.

The initial retrograde bougienage with passage of a thread was performed April 6, forty-one days after the esophagoscopy, and he was discharged April 9. Dilatations were continued over the thread at weekly intervals for several months until the esophagus had a diameter of a No. 45 French (1.5 cm.) bougie, and he was taking a normal diet without restriction or discomfort. The gastrostomy tube was removed, and he has continued well, receiving interval dilatations to maintain a satisfactory lumen through the stricture.

CASE 2.—A 29-year-old woman was admitted to the clinic Sept. 30, 1943, with cicatricial obliteration of the mid-thoracic esophagus of seven years' duration. The stricture was caused by the swallowing of a corrosive solution, and since that episode she had been unable to take even liquid nourishment by mouth. A gastrostomy stoma established shortly after the misadventure had furnished a means of providing adequate nutrition.

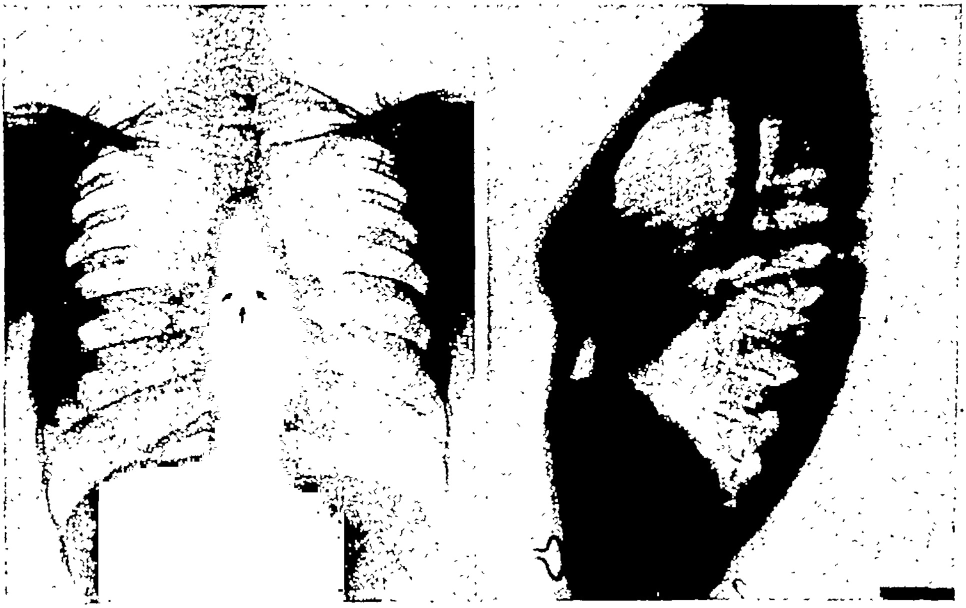


Fig. 1.

The findings of the general physical and laboratory examinations were within normal limits. Complete occlusion of the mid-thoracic esophagus 3 cm. below the aortic arch was demonstrated by fluoroscopy, roentgenograms with barium in the upper esophagus, and air insufflated into the lower esophagus through the gastrostomy opening (Fig. 1). Peroral esophagoscopy showed partial cicatricial contracture at the cricopharyngeus muscle, and the mid-esophagus ended abruptly in a cord of scar tissue without a lumen. By retrograde esophagoscopy, fibrous narrowing below the mid-esophageal stenosis was seen. These studies confirmed the diagnosis of fibrous obliterative stricture of the mid-esophagus, 2 cm. in length.

October 6, the stricture in the esophagus was widely exposed through a right transpleural and mediastinotomy incision. An esophagoscope was advanced to the stricture from above and another esophagoscope as near to its lower end as possible through the narrowed esophagus. The endoscopist introduced from below an aspirator used as a probe and exerted gentle pressure upon it, while the surgeon held the fibrous occluded esophagus between his fingers and guided the probe through its central portion. The task of traversing the 2 cm. stricture was most difficult and consumed more than one hour, but gradually a new lumen was made without perforation. After the aspirator was through the stricture, it was held steady between the surgeon's fingers. The endoscopist used a stream of saline to wash a silk thread down the upper esophagoscope. The thread was drawn into the aspirator by suction and the aspirator was pulled back within the lower esophagoscope.

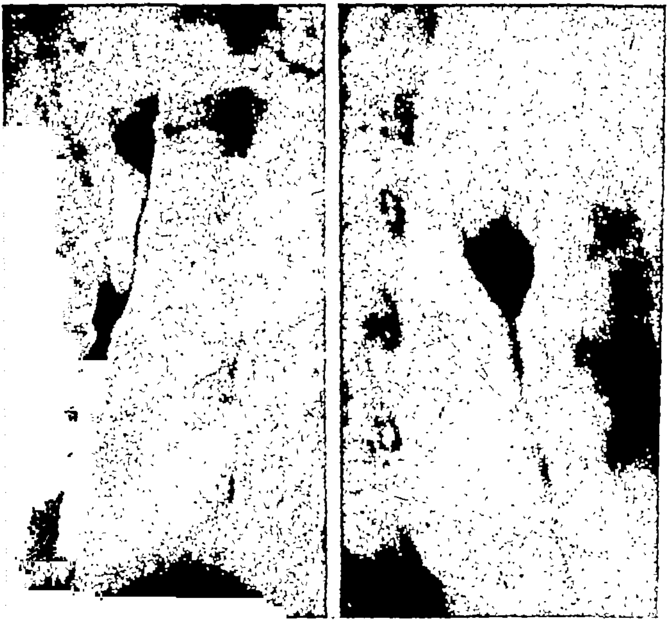


FIG. 2.

While the endoscopist and surgeon were concentrating upon the removal of the upper esophagoscope with retention of the thread through the stricture, the lower esophagoscope and aspirator were held by an attendant. Inadvertently the lower esophagoscope was allowed to slip backward into the stomach, and the end of the aspirator penetrated the esophagus 2 cm. above the cardiac orifice but several centimeters distal to the lower margin of the stricture. The injury to the esophageal wall was only the diameter of the aspirator, and it was immediately closed. This injury to the normal wall of the esophagus, after the successful execution of the preoperative plan for passage of the stricture and because of momentary diversion of personal surgical and endoscopic attention from a minor technical detail, was most exasperating.

Sulfanilamide was placed about the esophagus and a drain was carried from the mediastinum out through the chest wall incision. This patient also recovered.

November 10, thirty-five days after the operation, retrograde dilatation with Tucker bougies was begun. Gradually the lumen of the esophagus was increased until the patient could take a normal peroral diet. January 20, 1944, fluoroscopy with barium and roentgenograms showed the esophagus to have a lumen with mobile walls, and vigorous peristaltic waves passed along its entire course including the area of stricture (Fig. 2). The patient returned shortly afterward to her home in Trinidad, taking with her a set of Tucker retrograde bougies, with which dilatations have been continued by her local physician. When last seen at the clinic the stricture had a diameter of 9 mm. By report from the patient, progress has continued satisfactorily. The gastrostomy eventually will be closed.

DISCUSSION

An ingested corrosive solution is likely to cause stricture formation at one or more of four narrow points along the course of the esophagus. These are at the upper sphincter or cricopharyngeus muscle, at the crossing of the esophagus by the aortic arch, at the passage of the esophagus under the left primary bronchus, and at the lower sphincter or cardia. Observation of stricture cases indicates that the transient physiologic delay of food masses occurring at these locations makes possible increased local tissue injury when the swallowed material is a strong chemical irritant.

In Case 1 the stricture was at the aortic arch. In Case 2 it was near the left primary bronchus, although there was partial stricture formation both at the cricopharyngeus muscle and at the aortic arch.

Beyond the appropriate general treatment, the most important point for the physician to keep in mind is the prevention of complete esophageal obstruction in the early management of chemical injury of the esophagus. This is possible by having the patient swallow a thread promptly when there is any difficulty in taking liquids. This maintains a potential lumen even after extensive corrosive injury and also furnishes the essential guide for later dilatation. Passage of the thread through the intestinal tract allows adequate anchorage, and the portion within the esophagus should be kept strong and fresh by providing a constant supply from a bobbin. Should a gastrostomy be needed for feeding purposes, the thread may be brought out through that opening. With such an indwelling thread as a guide, gentle dilatation may be started within a few weeks of injury. We have not had occasion to employ the method wherein flexible mercury-filled bougies are passed from the first day following injury. In both cases here described, the esophageal lumen had been lost completely at the site of stricture for want of an inlying thread.

Dilatation is most safely attempted over a guiding thread. Tortuous or extensive strictures usually can be more safely dilated by retrograde bougienage with Tucker bougies through a gastrostomy opening than with solid metal dilators from above even with a guiding thread. This in itself is adequate reason for gastrostomy even if it is not essential for temporary feeding purposes.

As the first patient could not pass a thread through the stricture by swallowing and refused to allow gastrostomy, it would seem better judgment by hindsight to have declined the responsibility for treating him than to be forced by events to accept responsibility for a perforation that might not have occurred under the regimen of treatment considered preferable. The perforation produced by peroral dilatation, we believe, might have been prevented had gastrostomy been performed and retrograde bougies passed.

The risk of fatality in the treatment of strictures such as occurred in Case 2 has been so high that many have remained untreated. We felt that it presented too hazardous a situation to attempt endoscopic procedures alone, because the stricture itself was completely fibrous and of considerable length, and because the portion of the esophagus below the obliterated area could not be well visualized because of narrowing. On the other hand, we were quite certain that gradual dilatation would in time permit satisfactory resumption of function if the strictured area could be canalized without causing a fatal mediastinal infection. This opinion is based upon our experience with the previously mentioned cases of esophageal stricture from various causes in which treatment by repeated dilatations was most satisfactory.

In the absence of an indwelling thread or endoscopic means of safely passing one, the possible alternatives were debated. The most obvious solution was to refrain from any treatment and let an otherwise healthy woman of 29 years attain life expectancy on gastrostomy feeding. The patient was more than willing to take risks to regain the privilege of eating by mouth. An external esophagoplasty was considered and discarded after analogy with this procedure for carcinoma of the esophagus in which several operative stages produce an arrangement devoid of cosmetic appeal and rarely resulting in perfect function al-which was at the introitus, was cut between clamps and ligated. There was no other attachment of the tumor to the wall of the esophagus (Fig. 2). The tumor was removed completely. The esophagus was then closed with one continuous silk suture and two rows of interrupted silk

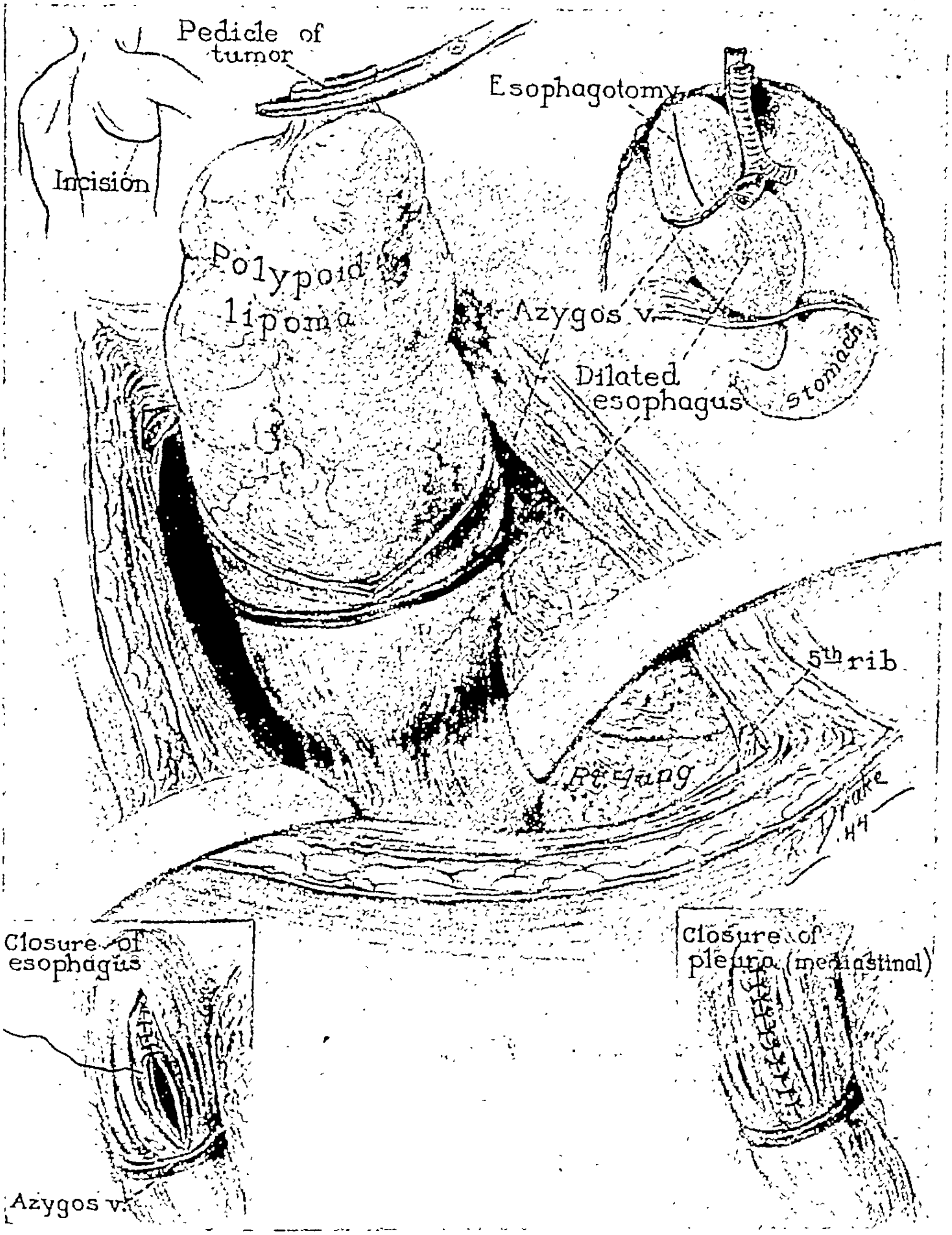
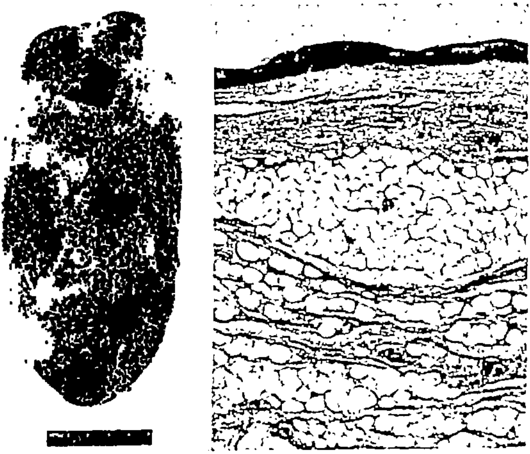


Fig. 2.—Transpleural esophagotomy for complete removal of pedunculated polypoid lipoma which was attached at the introitus of the esophagus (Case 1). The tumor caused a marked dilatation and displacement of the esophagus, filled the entire lumen and extended to the cardia. Left insert shows posterolateral incision. Upper right insert shows marked displacement and dilatation of the esophagus and the site of the incision of the esophagus above the azygos vein. Lower left insert shows closure of esophagus by using a continuous inverting suture of silk. Lower right insert shows the completion of the closure of the esophagus and overlying mediastinal pleura with interrupted silk sutures.

sutures. The mediastinal pleura was sutured over the line of closure in the esophagus. The pleural cavity was irrigated with saline solution and 10 Gm. of sulfathiazole were placed in the pleural cavity. The lung was inflated with positive pressure and closed drainage of the pleural cavity was instituted through the periosteum of the fifth rib, anteriorly.

The tumor was 22 cm. long and 8.5 by 5.5 cm. wide. It was covered with normal mucosa. On microscopic examination it was found to be a polypoid lipoma (Fig. 3, *A* and *B*).



A. *B.*

Fig. 3.—*A*, Large polypoid lipoma covered with normal esophageal mucosa which is ulcerated in places (Case 1); *B*, section of tumor (×32).

The patient's immediate convalescence was very satisfactory. On the third day his temperature was 101° F. and his pulse rate increased. The leucocyte count was 9,700. The next day his temperature was 103° F. and the leucocyte count was 9,000. There was only moderate drainage from the pleural cavity through the closed drainage tube. A culture of the material draining from the pleural cavity revealed green-producing streptococci. Penicillin therapy was employed. His temperature gradually dropped to 99° F. on the eighth day, and at this time open drainage of the empyema on the right side was instituted. There was a moderate-sized cavity which gradually closed.

Roentgenograms made at the time of the patient's dismissal, March 27, revealed considerable reduction in the size of the esophagus but it was still moderately dilated. The right lung had re-expanded and the empyema cavity had become completely obliterated. His symptoms had disappeared completely, he had gained twenty pounds (9.1 kg.), and his general condition was very satisfactory.

CASE 2.—A man, aged 24 years, came to the clinic Aug. 7, 1940, complaining chiefly of sore throat, dysphagia, cough, pain in the thorax, and weakness. He had been perfectly well until three and one-half years previously, when he had had an acute infection of the upper respiratory tract and a severe sore throat which had persisted since that time. Difficulty in swallowing had begun about the same time. It had occurred periodically and often had disappeared for a week or more. He had had several attacks, during which he had been unable to swallow any food for a day or more at a time, and he always had had to wash his food down with large quantities of water. He had had a cough which had been productive of clear mucus which had seemed to accumulate in his throat. This had been more severe when his throat had been very sore. At times, he had noticed bloodstreaked sputum. There was a questionable history of melena. He had had attacks of vomiting which had been associated with a choking sensation in his throat and retrosternal pain. On four occasions a mass had been coughed up and nearly had strangled him when it had protruded from his mouth. This first had occurred in the course of a vomiting spell. He had attempted to bite the tumor off but had been unable to do so. He had replaced it in his mouth and had swallowed it. Regurgitation of the tumor had not occurred recently but he had had increasing difficulty with regurgitation of food, progressive loss of weight, and weakness. He had had several examinations both roentgenologically and esophagoscopically before he came to the clinic and it had been thought that his condition was due to cardiospasm. The presence of a tumor had not been recognized.

At the time of his visit to the clinic, his general condition was rather good. He had lost about fifteen pounds (6.8 kg.). The value for the hemoglobin was 6.6 Gm. per 100 c.c. of blood. The erythrocytes numbered 4,080,000 and leucocytes 8,200 per cubic millimeter of blood, respectively. Roentgenologic examination of the esophagus revealed severe dilatation with lagging at the cardia. A diagnosis of cardiospasm was made (Fig. 4). Examination of the stomach did not disclose any abnormality. Esophagoscopic examination revealed a large pedunculated esophageal tumor which was attached to the posterior wall of the esophagus just below the cricoid cartilage. The tumor extended down into the middle third of the esophagus. An attempt was made to bring the tumor out of the esophagus but this was impossible because the tumor was tightly wedged into the esophagus. In addition, the patient had an associated cardiospasm. Operation was necessary not only because of the presence of a tumor but because it was producing progressive dysphagia which resulted in an inability to obtain sufficient nourishment. Because the pedicle of the tumor was found to be in the introitus and the tumor was of moderate size, it was decided that cervical esophagotomy would be the best method of approach for removal of the tumor.

Aug. 14, 1940, one of us (S. W. H.) performed a cervical esophagotomy by approaching the tumor from the left cervical region. An incision was made along the anterior border of the left sternomastoid muscle. The carotid and jugular vessels were retracted to the left, and the left inferior thyroid artery and vein were ligated. Elevation of the thyroid gland exposed the esophagus, which was markedly dilated and edematous. A longitudinal incision was made into the lateral wall of the esophagus. The esophagus was explored and it was found that there were two tumors, each with a pedicle originating from a single pedicle in the introitus of the esophagus (Fig. 5). The single pedicle of origin was cut between clamps and ligated. The tumor was completely removed through the esophagotomy wound. The incision in the lateral wall of the esophagus was closed with one layer of interrupted catgut sutures, which approximated the mucosa, and with two layers of interrupted silk sutures which closed the wall of the esophagus.

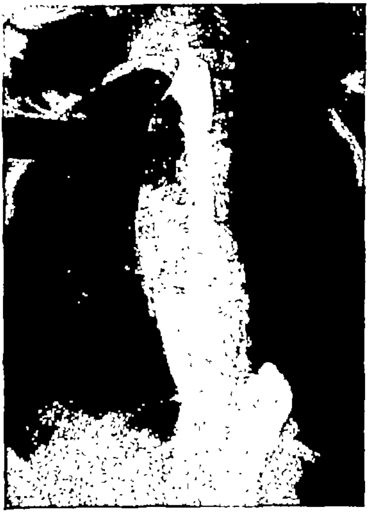


Fig. 4.—Dilatation of esophagus originally thought to be due to cardiospasm (Case 2): esophagoscopy revealed a pedunculated tumor of the esophagus.

One tumor measured 10 by 5.5 cm. and the other 7.5 by 3.5 cm. The two tumors had a pedicle 6 cm. in length and were attached to the esophagus by one pedicle (Fig. 6 *A* and *B*). Microscopic examination disclosed that the tumors were myxofibromas. They were covered with normal esophageal mucosa which was ulcerated over much of the surface of each tumor. This undoubtedly accounted for the bleeding which had occurred and also for the secondary anemia which had been present before operation.

The convalescence was uneventful and the patient was dismissed Sept. 16, 1940, at which time the wound had healed entirely. His general condition was very good and he had been entirely relieved of symptoms. In a recent letter, he stated that he had not had any recurrence of his former symptoms.

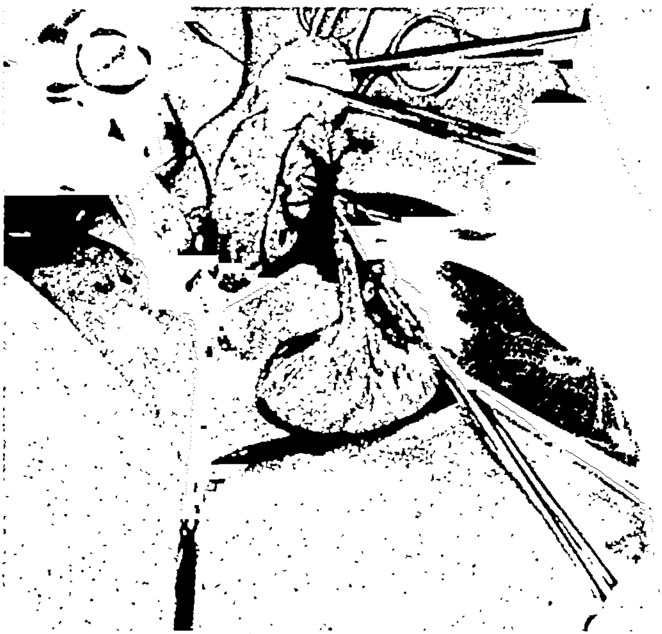
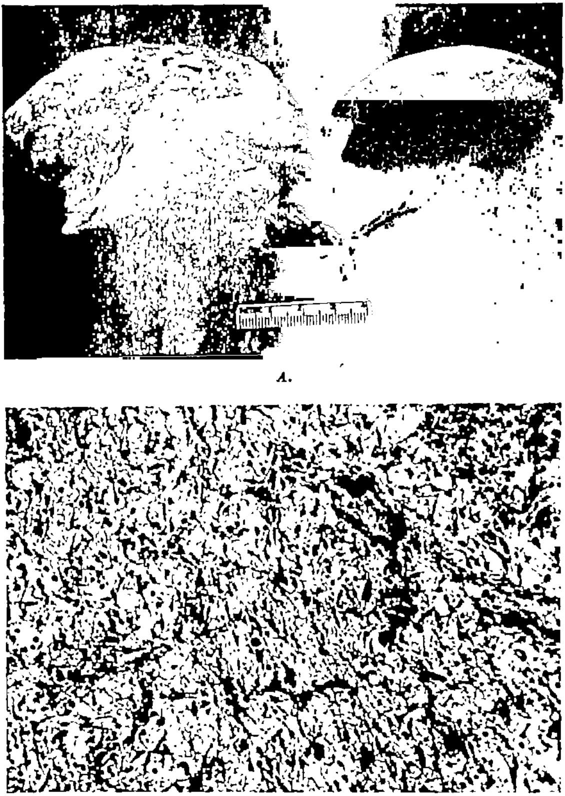


Fig. 5.—Left cervical esophagotomy for removal of two pedunculated tumors (myxofibromas) originating from a single pedicle at the esophageal introitus (Case 2).

Case 3.—A woman, aged 56 years, came to the clinic May 17, 1943. Her chief complaint was pain beneath the xyphoid, of one month's duration. She had not had any symptoms prior to one month previously, when she had noticed pain beneath the lower part of the sternum and xyphoid cartilage, which had been moderately severe. The pain, which had been localized to this region, had lasted from five to ten minutes. It often had been present when she had arisen in the morning and it had been present at irregular intervals throughout the day. Heat on some occasions had relieved the pain. Food or swallowing had not had any effect on producing or relieving the pain. She had not had any difficulty in swallowing solid food and had not regurgitated her food. She frequently had been annoyed by a gagging sensation which had been associated with regurgitation of a small amount of mucus. She had been treated for anemia for three years and there had been some suspicion that the anemia was of a pernicious type but this diagnosis had not been established definitely. The anemia always had responded well to treatment with liver extract.

The patient weighed 133 pounds (60.3 kg.), which represented a loss of ten pounds (4.5 kg.). The blood pressure was 180 mm. of mercury, systolic, and 95 mm. diastolic. The value for the hemoglobin was 13.1 Gm. per 100 c.c. of blood. The erythrocyte count was 5,030,000 and leucocyte count was 8,500. A roentgenogram of her thorax revealed a tumor mass lying to the right and behind the lower part of the esophagus and cardia of the stomach. The mass apparently extended through the


A.

B.

Fig. 6.—*A*, Pedunculated tumors arising from the esophageal introitus (Case 2); *B*, section of one of the tumors showing a myxofibromatous structure (×125).

esophageal hiatus. A roentgenogram of the esophagus and stomach showed a lesion involving the distal portion of the esophagus and the cardia (Fig. 7). There was a large extrinsic mass which appeared to lie sometimes in the thorax and sometimes in the abdomen, as though it slipped back and forth through the hiatus. There also was a duodenal ulcer with a shallow crater. Esophagoscopic examination revealed an obstruction at the lower end of the esophagus owing to a tumor which involved the wall of the esophagus and pushed the esophagus posteriorly and to the left. The mucous membrane of the esophagus was normal and intact and there was no lesion of the cardia of the stomach. A diagnosis of a probable intramural leiomyoma was made. Although there was no history of dysphagia, operation was advised because of the large tumor which was disclosed by roentgenography and because of dilatation of the esophagus above the tumor. An abdominal approach was employed because a considerable portion of the tumor protruded through the hiatus into the abdomen.

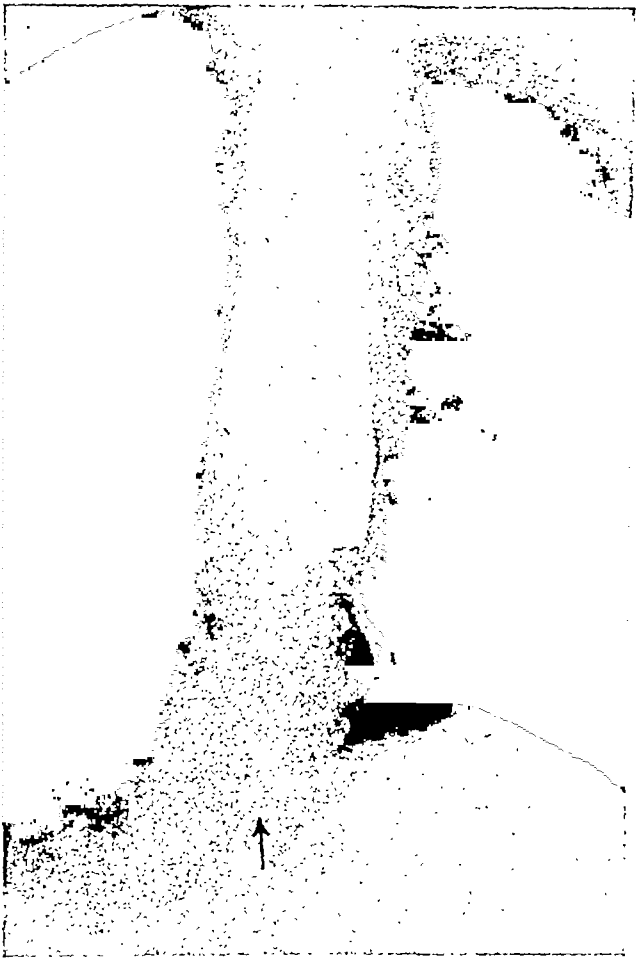


Fig. 7.—Esophageal tumor extending through esophageal hiatus and obstructing lower part of the esophagus at the cardia (Case 3).

May 13, 1943, operation was performed by one of us (S. W. H.). A left rectus incision was employed. A large leiomyoma was found involving the lower part of the esophagus; the greater portion of the tumor was in the posterior mediastinum and extended through the hiatus into the abdomen (Fig. 8). The tumor was removed and the opening in the lower part of the esophagus and cardiac end of the stomach was repaired with interrupted silk sutures. A small portion of the tumor, which extended deep into the posterior mediastinum, was not removed because of its fixation to the pericardium. It was felt that this would undergo atrophy. If not, it could be removed later. Microscopic examination revealed a leiomyoma. The tumor measured 15 by 7 cm. and weighed 190 Gm. (Fig. 9, *A* and *B*). The patient's convalescence was uneventful.

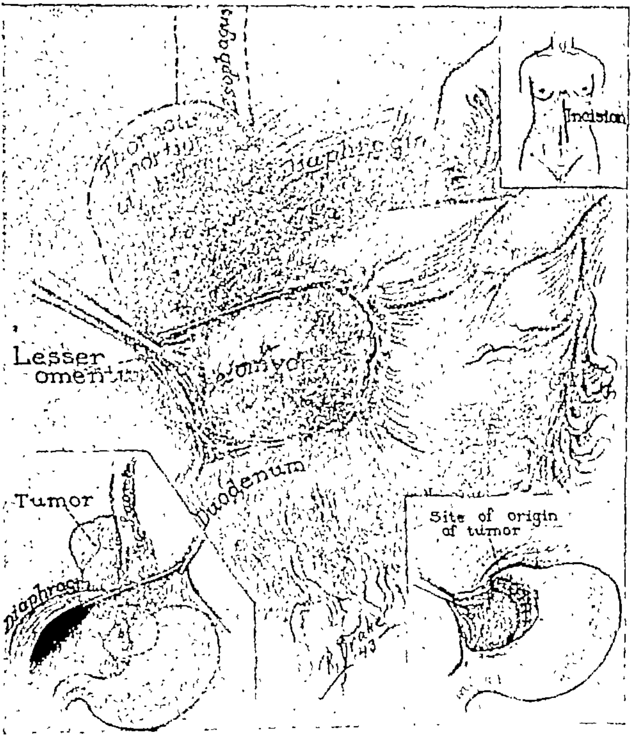


Fig. 8.—Large leiomyoma of the right wall of the lower part of esophagus and cardia extending through and enlarging the esophageal hiatus and partially obstructing the esophagus at the cardia (Case 3). Tumor was removed through an oblique left rectus incision. Esophagus and cardia were repaired with interrupted silk sutures.

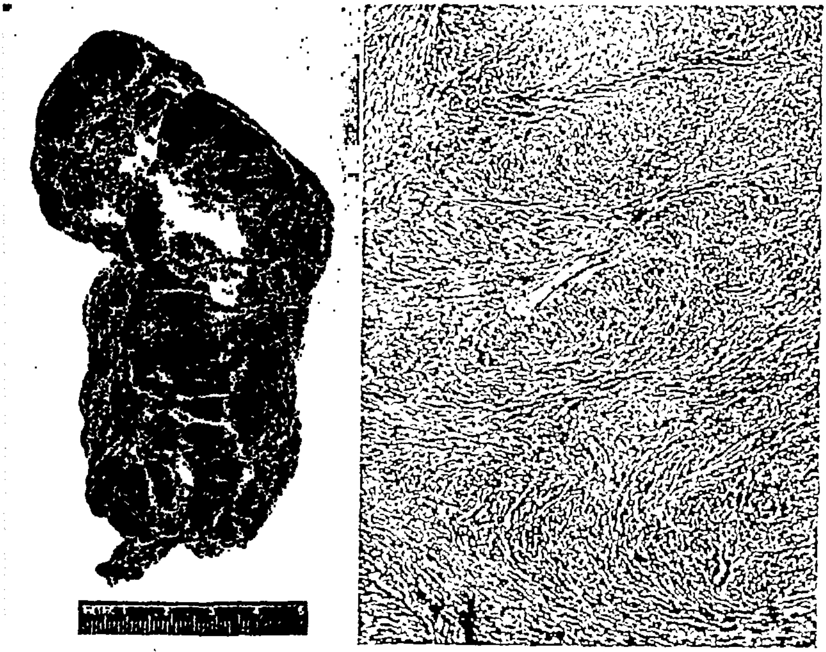

A. B.

Fig. 9.—*A*, Large leiomyoma removed from lower part of esophagus (Case 3); *B*, section of tumor (×70).

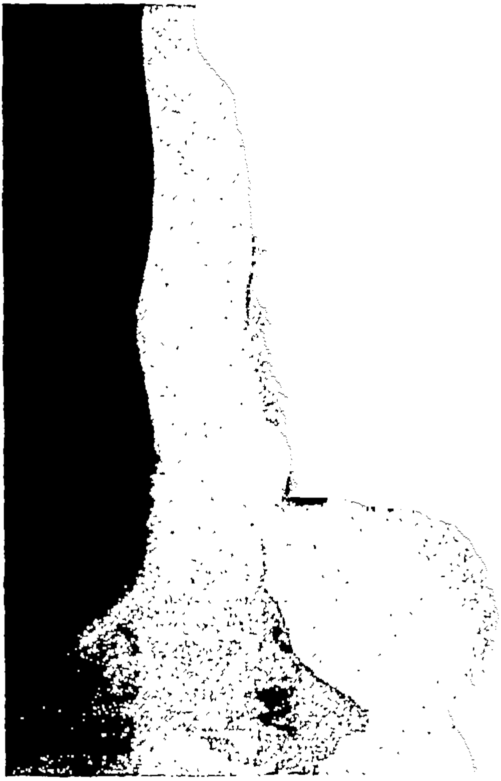


Fig. 10.—Roentgenogram made five months after operation in Case 3; esophagus is normal. (Small esophageal hiatus hernia resulting from the enlargement of the esophageal hiatus by the tumor.)

She was dismissed from our care June 21, 1943, at which time the wound had healed entirely and her general condition was very good. A roentgenogram showed slight deformity of the lower end of the esophagus (Fig. 10). She had no subjective symptoms. She was advised to return for observation in three months. When she returned, Oct. 26, 1943, she had no subjective symptoms. Her appetite had been very good. She had had no dysphagia. As her blood pressure had increased to 210 mm. of mercury, treatment for hypertension was instituted. A roentgenogram showed a very slight deformity of the lower end of the esophagus which was much less pronounced than it had been at the time of her dismissal; this indicated atrophy of the residual portion of the tumor in the posterior mediastinum. In view of the fact that her progress has been very satisfactory, we do not believe it will be necessary to remove the remaining portion of the tumor; however, she will be kept under observation.

CASE 4.—A woman, aged 39 years, came to the clinic March 8, 1943, complaining of cough of fifteen years' duration. During this time she had had periodic attacks of cough in the winter. The cough had been continuous throughout the cold weather but had been much less noticeable in the summer. It had been relatively nonproductive until about three months before she came to the clinic, when she had had severe colds associated with chills, fever, and expectoration of purulent material. Since that time she had had a constant hacking cough with moderate dyspnea, and a short time before she came to the clinic she had noticed some diffuse substernal pain on swallowing. She had not had any dysphagia.

When the patient was examined at the clinic, the systolic blood pressure was 105 mm. of mercury and the diastolic pressure was 74 mm. A roentgenogram of her thorax showed that the lung fields were essentially normal. There was a large rounded soft tumor mass in the posterior mediastinum. There was some compression of both main bronchi and some widening of the angle of the coryna. A roentgenogram showed a tumor, approximately 5 by 8 cm., lying posterior and to the right of the middle third of the esophagus (Fig. 11, *A* and *B*). The tumor was compressing the lumen of the esophagus and probably had originated in the wall of the esophagus. On bronchoscopy, the coryna was found to be greatly flattened. The right main bronchus was displaced laterally and the lumen was reduced to approximately one-half its normal diameter owing to pressure on the medial wall. The left main bronchus was displaced further to the left than normal. Esophagoscopy revealed that the esophagus was displaced to the right by a mass at the level of the middle third. The mucous membrane was intact.

March 7, a right transpleural mediastinotomy was performed by one of us (S. W. H.). A posterolateral incision was made and the posterior two-thirds of the fifth rib were resected. The pleural cavity was opened through the posterior periosteum of this rib and a large tumor was found in the anterior wall of the esophagus. The tumor was very adherent to the right and anterior portions of the pericardium. It projected between the two main bronchi, to which it was very adherent. The right bronchus had been compressed more than had the left bronchus. It was difficult to determine the site of the esophagus posteriorly. A large stomach tube was passed through the esophagus to determine its position.

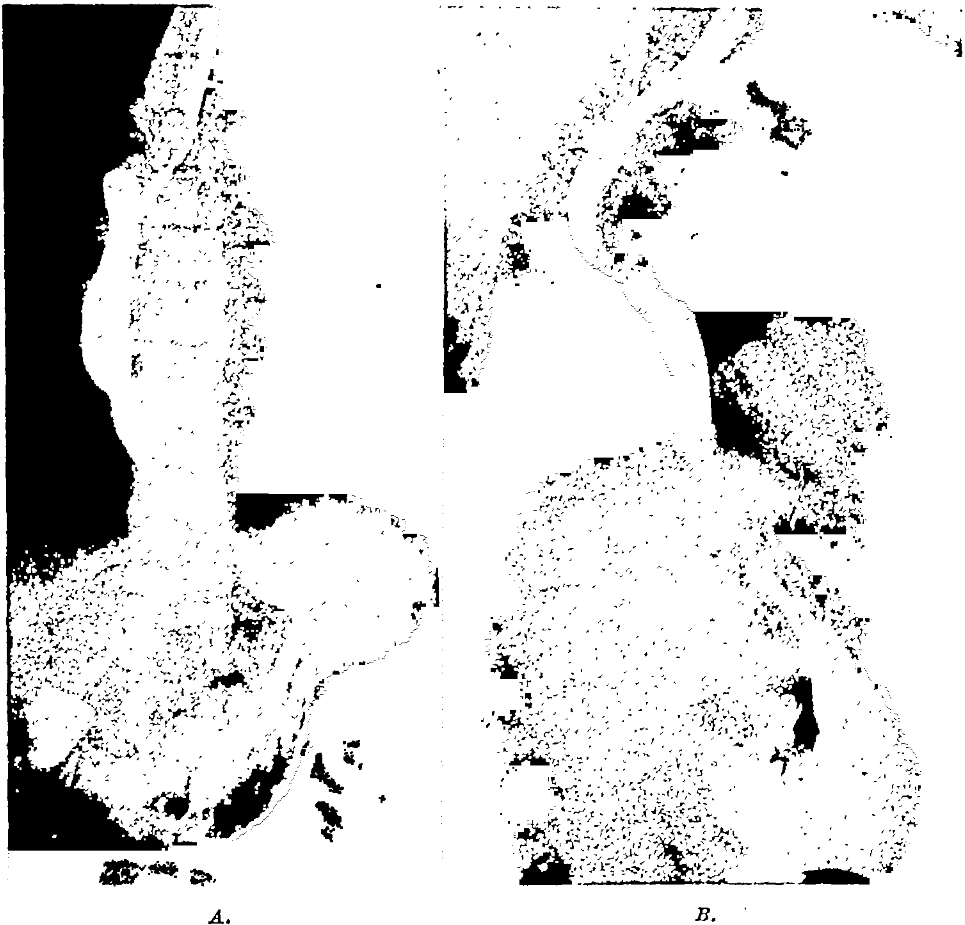

A. *B.*

Fig. 11.—Tumor of anterior wall of the middle third of the esophagus (Case 4); tumor is displacing the esophagus to the right and posteriorly; *A*, anteroposterior view; *B*, lateral view.

The tumor was very adherent to all of the surrounding structures. It was removed completely (Fig. 12). The tumor was very friable and degenerated in the central portion. There was some necrosis of tissue. The tumor contained about six ounces (180 c.c.) of thick necrotic puslike material. The esophagus was reinforced with interrupted silk sutures at the site of removal of the tumor. The lumen of the esophagus was not opened. The thoracic cavity was irrigated with physiologic salt solution. Ten grams of sulfathiazole were placed in the pleural cavity, which was drained with a 24 F. catheter. Microscopic study of the tissue revealed an infected cyst that was lined with glandular epithelium and contained smooth muscle in its wall.

The patient made an uneventful convalescence. The highest temperature was 100° F. on the fourth day. The temperature then dropped to normal and remained normal until her dismissal on the twenty-third

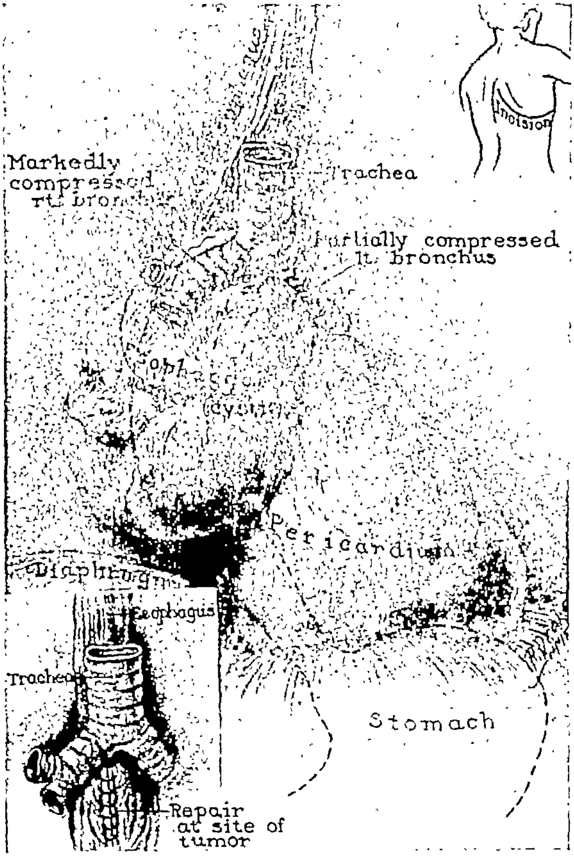


Fig. 12.—Cystic tumor originating from the anterior wall of the middle third of the esophagus (Case 4), displacing the esophagus to the right (and posteriorly), causing marked compression of the right main bronchus, moderate compression of the left main bronchus, and widening of the corynal angle. Tumor is adherent to the right portion of the pericardium. Upper right insert shows posterolateral incision for transpleural removal of tumor. Left lower insert shows repair of esophagus at the site of the tumor.

day, at which time the wound had healed entirely. She had been relieved entirely of the cough and had no subjective symptoms. Roentgenograms of the thorax were essentially normal at this time. When she returned for observation six months after operation, her general condition was very good. She had not had any recurrence of dyspnea. She stated that occasionally dry food would stick in the upper part of her esophagus. Esophagoscopic examination revealed a slight herniation of the esophagus wall at the point of removal of the tumor and slight narrowing of the esophagus below this region.

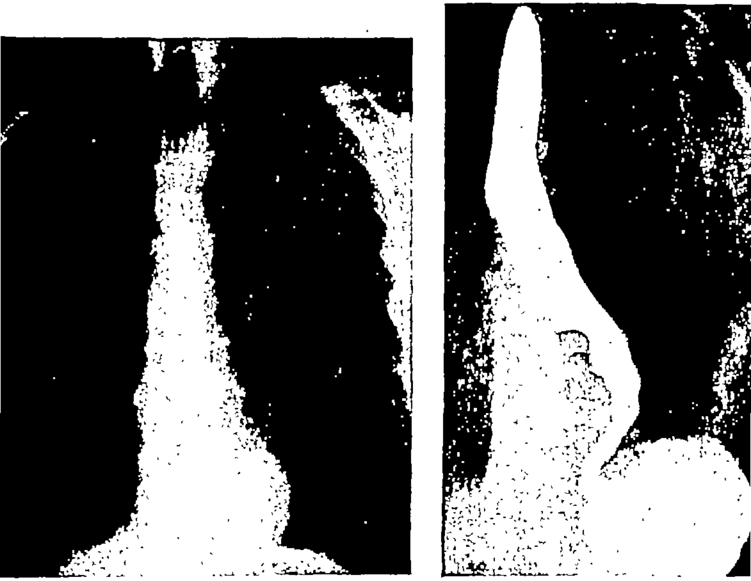
CASE 5.—A man, aged 39 years, came to the clinic May 18, 1942, complaining of asthma, bronchitis, hay fever, nervousness, and fatigue, of four months' duration. He had had asthmatic bronchitis and hay fever for twenty years. About four months before he came to the clinic he had begun to feel extremely tired and had become fatigued after work; he had had dizzy spells, particularly when walking, and had had sensations of falling and many other nervous symptoms.

The results of physical examination were essentially negative except for emphysema and râles throughout both lungs. The results of all laboratory tests were essentially negative except that roentgenologic examination of the thorax revealed some emphysema and a shadow of increased density behind and a little to the left of the heart (Fig. 13*A*). It was felt that this probably was due to a diaphragmatic hernia. A roentgenogram of the stomach and esophagus revealed a deformity of the lower end of the esophagus just above the cardia (Fig. 13*B*). The patient had not had any dysphagia.

Esophagoscopic examination showed a marked deformity of the lower end of the esophagus which extended to a point fourteen and one-half inches (40.6 cm.) from the incisor teeth. The esophagus was angulated to the left. There was no obstruction at the cardia and the action of the sphincter seemed to be gone. There was some irritation of the mucous membrane owing to regurgitation of gastric secretion. The mucous membrane was intact. There was no indication of a tumor which would permit biopsy. Because of the many general symptoms and relatively poor general condition of the patient, neither of which was related to the esophageal tumor which had been discovered in the course of a previous examination, operation was not advised. General supportive treatment was employed for the general symptoms.

When the patient returned to the clinic after an absence of three months, his general condition was somewhat improved and he had no symptoms referable to the tumor, which had not changed in size. He was advised to continue with the general treatment. When he returned again, May 2, 1943, his general condition had improved considerably but he was greatly worried over the presence of the tumor. Complete examination, including roentgenographic studies (Fig. 13*B*) and esophagoscopy, disclosed no appreciable change in the size of the tumor;

however, because of his anxiety in regard to its presence and owing to the fact that his general condition had improved, we felt justified in advising surgical treatment. At this time, the value for the hemoglobin was 17 Gm. per 100 c.c. of blood. The erythrocyte count was 4,446,000 and the leucocyte count was 8,600. The value for the blood urea was normal. Urinalysis did not reveal any abnormality.



A. *B.*

Fig. 13.—*A*, Roentgenogram of thorax, made when the patient in Case 3 first came to the clinic, showing emphysema of lungs and increased density behind the cardiac shadow; *B*, a subsequent roentgenogram disclosed that this increased density was due to a tumor of the lower part of the esophagus. One year later, when the patient returned to the clinic, the appearance of the esophagus in the roentgenogram was the same as it was in *B*.

June 12, 1943, the tumor was exposed by a left transpleural incision. The lower fourth (17.8 cm.) of the esophagus and the cardiac end of the stomach were resected. A left posterolateral incision was made and a portion of the eighth rib was resected. The tumor involved the entire wall of the lower 5 inches (12.7 cm.) of the esophagus. It extended down to the cardia of the stomach. The tumor was very lobulated and the structure of the wall of the esophagus was not normal. It was necessary to make a transdiaphragmatic incision at the lower end of the tumor at the cardia. The upper end of the stomach was closed and the esophagus was anastomosed to the anterior wall of the stomach. The cardiac end of the stomach was transposed into the thoracic cavity and an esophagogastrostomy was performed (Fig. 14).

Microscopic examination revealed a leiomyoma which completely surrounded and compressed the esophagus (Fig. 15).

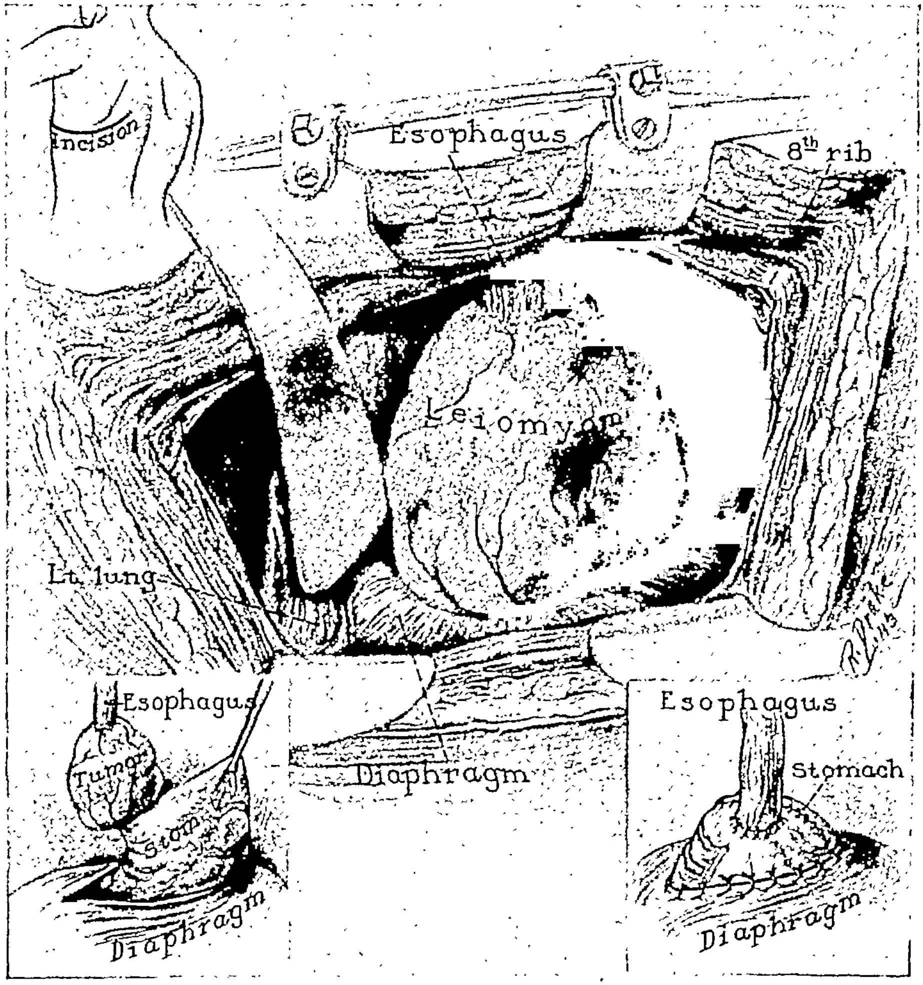


Fig. 14.—Resection of lower fourth of esophagus for removal of leiomyoma in Case 5. The stomach was transposed into the thoracic cavity (lower left insert), and the esophagus was anastomosed to the anterior wall of the stomach (lower right insert).

The immediate response to the operation was good except for an increase in the respiratory rate. The patient immediately was placed in an oxygen tent. His temperature gradually rose to 103° F. and there was an increase in his respiratory rate as well as an increase in his pulse rate. His condition became gradually worse and he died on the fourth day, of bilateral bronchopneumonia.

This is the only death which has occurred in removal of benign tumors of the esophagus and we do not believe this death was due as much to the operative procedure as it was to the previous pulmonary condition.

The patient had had asthmatic bronchiectasis for twenty years and we believe it is questionable whether or not he should have been subjected to surgical treatment for two reasons: first, because he had been refused operation on two previous occasions, and second, because of his poor general condition as well as the fact that we did not believe the tumor was responsible for his symptoms.

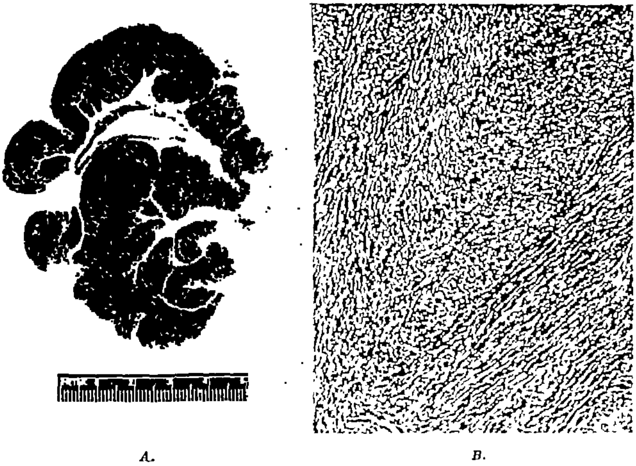


Fig. 15.—Intramural leiomyoma involving entire wall of esophagus but not involving mucous membrane; *A*, gross appearance; *B*, section of tumor (×70).

CASE 6.—A man, aged 62 years, came to the clinic Nov. 20, 1921. Six years previously, he had coughed and raised into his mouth what seemed to be a small growth attached to the palate; he had swallowed the growth again. His throat had been swollen and sore for five days thereafter. He had thought that the growth was an enlarged palate. Three weeks before he came to the clinic, he had eaten too much and had vomited and brought up a piece of tissue long enough to protrude from his mouth. He again had swallowed the tissue which was the size of his little finger. Two weeks before he came to the clinic he had experienced the same thing on two occasions and had felt a tickling sensation in his throat. He had had no difficulty in swallowing.

An esophagoscopic examination revealed a long, narrow, pedunculated tumor extending down the esophagus. The tumor was situated on the right wall of the esophagus about one and one-half inch (3.8 cm.) below the introitus. No attempt was made to pull it up.

Dec. 7, 1921, Dr. E. S. Judd operated upon the patient through a left cervical incision in the anterior border of the sternomastoid muscle. Esophagotomy was performed and the tumor was excised at the base of

Great interest is also attached to the follow-up study of the operative survivors (Table V). In the adenocarcinoma group, 4 died of recurrence of the disease on the average of one year later. One patient died of cancer of the breast with pulmonary metastases three years after esophageal resection. Ten patients were alive and apparently free of disease from four years to six weeks. In the group of 16 patients with squamous cell cancers, 8 died of recurrence after periods varying from two years to nine months. Curiously enough, nearly every one had had a Torek operation. One patient died of coronary thrombosis three and one-half months later. Seven patients were alive and well from eight years to one month. In the entire series, therefore, there are 17 patients who are alive and apparently free of disease at the time of this communication.

This study, which has been carried on over a period of eight years, has clarified many aspects of the diagnosis and treatment of this disease and I would like to enumerate some of the important points which deserve emphasis:

1. We must forget what the old textbooks tell us that cancer of the esophagus is usually seen in the seventh and eighth decades. I have seen a few in the third and many in the fourth.

2. Duration of the symptoms of dysphagia cannot be used as a criterion of operability. I have noted an inoperable tumor with a six weeks' history and an operable one with one year's history. Operability is determined more by the biologic characteristics of the individual tumor as evidenced by grade of malignancy, invasiveness, and rapidity of spread to regional and distant lymph nodes.

3. Boring pain in the back has serious connotations and usually indicates tumor fixation posteriorly. This may or may not be accompanied by local shock tenderness.

4. The extent of weight loss is a variable factor. Those tumors which impinge early on the esophageal lumen cause rapid weight loss, a considerable part of which may be due to dehydration. Some patients with extreme weight loss have regained a pound a day during the preoperative period by parenteral hydration and were found to have removable growths. Others with minimal or no weight loss have presented inoperable tumors by reason of the fact that growth was in a peripheral direction.

5. The finding of a smooth conical obstruction at the lower end of the esophagus in the x-ray picture does not necessarily indicate a benign cardiospasm. Esophagoscopy must be carried out. Negative esophagoscopic findings do not exclude completely the presence of a cardial carcinoma. Repeated endoscopic examinations are indicated if the symptoms persist.

6. No patient should be subjected to operation unless the biopsy specimen obtained by esophagoscopy shows carcinoma.

7. The finding of squamous cell tumor in the biopsy specimen indicates origin in the esophagus. When adenocarcinoma or mucous cell carcinoma is reported, the evidence is clear that the tumor originated at the cardiac end of the stomach. I have never encountered an instance of carcinomatous degeneration of misplaced gastric mucosa in the esophagus. Every case of adenocarcinoma of the esophagus, no matter how far up the organ, has been found to have its origin at the cardia. It is important to determine the histologic characteristics of the tumor because the surgical approach is different in each instance.

8. Every patient who is a candidate for radical surgery of the esophagus must have a careful appraisal of his physical and physiologic status. This should include a thorough physical examination, electrocardiography, vital capacity determination, renal function studies, and careful attention to the condition of the teeth and gums.

9. The importance of a careful rectal examination in cases of adenocarcinoma of the cardia cannot be overemphasized. The finding of a rectal shelf indicates inoperability. The presence of enlarged firm lymph nodes in the neck, especially in the supraclavicular region, demands biopsy to exclude peripheral metastases. Hoarseness with cancers of the middle third calls for laryngeal examination. Paralysis of the left recurrent nerve indicates invasion by tumor and probable inoperability.

10. In the preoperative preparatory period, the value of intravenous hydration by saline and glucose, plasma, whole blood, and amino acids has been amply demonstrated.

11. The necessity of a preliminary gastrostomy has been completely obviated because the presence of a gastrostomy interferes with mobilization of the stomach in the performance of an intrathoracic anastomosis. Should adjuvant alimentary feeding be indicated, jejunostomy is more logical and preferable.

12. The need of an expert anesthetist for this operation requires no emphasis. We have used a combination of avertin and controlled positive pressure ethylene and ether inhalation anesthesia, and have not been impressed with any superior virtues of intratracheal anesthesia.

13. There are many details of the operative management which merit emphasis. Complete hemostasis, avoidance of operative trauma, and unhurried manipulative procedures make for a smoother convalescence, decreased morbidity, and greater chance of recovery. In mobilizing the esophagus, great care should be exerted to avoid injury to the external musculature of the organ. Any accidental tear should be carefully repaired with fine silk. Otherwise a spontaneous perforation may occur postoperatively. The growth may be found locally inoperable by reason of fixation to the aorta, vertebral column, or hilus of either lung. Invasion of the right mediastinal pleura does not necessarily indicate inoperability because it may be possible to excise part of this

pleura and bring the patient safely through the operative ordeal. There is no question but that the accidental opening of the right pleural cavity confronts the patient with a serious physiologic disturbance and greatly increases the risk of the operation. However, if tumor tissue must be excised, the surgeon has no other choice but to perform as radical an operation as is possible. The opening in the right pleura may be effectively closed by using the transplanted stomach, but the surgeon must not fail to aspirate the right side of the chest at the completion of the procedure in order to remove trapped air and thus prevent a tension pneumothorax. In mobilizing the stomach for transplantation, great care should be exerted to maintain an adequate blood supply. This is particularly applicable when an anastomosis above the aortic arch is contemplated. The same applies to the stump of the ablated esophagus, an organ which has a precarious blood supply at best. I want to stress particularly the need for a careful and accurate suture anastomosis between esophagus and stomach. This requires interrupted silk sutures, accurately placed and tied without tension, careful approximation of mucosa to mucosa and telescoping of stomach over the suture line to obviate tension and drag. We have not encountered one instance of leakage from the line of anastomosis and attribute this solely to the importance that we have attached to this part of the operation.

Another important point is the need for a careful repair of the diaphragm and fixation of its cut edge to the wall of the transplanted stomach to prevent herniation of bowel into the chest. The diaphragm should be put at rest by pinching the phrenic nerve. The local use of sulfonamides to minimize mediastinal and pleural infection has considerable merit. We have had no cause to regret the routine employment of underwater drainage of the pleural cavity in the postoperative period.

14. All patients are placed in an oxygen tent at the completion of the operation.

15. No swallowing is permitted for four or five days. If a careful anastomosis has been made, it is unnecessary to extend this for a longer period.

16. The surgeon must always be on the lookout for postoperative chest complications, such as encapsulated accumulations of air and fluid, infection of the pleural cavity, bronchial obstructions, etc. This requires daily chest examinations, bedside roentgenograms and clinical appraisal. There should be no hesitancy in employing bronchoscopy to relieve the patient of tenacious secretions. This is often a lifesaving procedure. The bedside x-ray picture may be misleading and the surgeon should, therefore, rely more on the clinical examination. Persistant dyspnea and excessive bronchial secretion usually indicate an accumulation of air or fluid or both and the surgeon should not delay aspiration.

17. Infections should be dealt with as soon as possible, because these debilitated patients cannot withstand infection for long.

It must be emphasized that the care of a patient with cancer of the esophagus is a cöoperative problem requiring the concerted efforts of roentgenologist, esophagoscopist, anesthetist, operating room staff, bedside nurses, physician, and surgeon. Even under the most ideal conditions, the operative mortality is relatively high if the surgeon does not practice careful selection of his operative material. But the situation today is far different than it was fifteen years ago. Marked progress has been made and with increasing experience, I look forward confidently to a generally lower mortality and a higher percentage of cures.

DISCUSSION ON "THE MANAGEMENT OF STRICTURE OF THE ESOPHAGUS" BY DR. RALPH ADAMS AND DR. WALTER B. HOOVER;* "SURGICAL TREATMENT AND CLINICAL MANIFESTATIONS OF BENIGN TUMORS OF THE ESOPHAGUS WITH REPORT OF SEVEN CASES" BY DR. STUART W. HARRINGTON AND DR. HERMAN J. MOERSCH;† AND "CAUSES OF MORTALITY FOLLOWING RADICAL RESECTION OF THE ESOPHAGUS FOR CARCINOMA" BY DR. JOHN H. GARLOCK.

DR. RICHARD H. SWEET, Boston, Mass.—It is not proper for me to take much of your time to discuss these three interesting reports. That by Dr. Harrington and Dr. Moersch is so far out of my experience that I shall say nothing about it.

I would like to comment, however, on the interesting and valuable reports by Dr. Adams and Dr. Hoover, and Dr. Garlock. First of all I would like to suggest for your consideration that it might not be quite proper for the former to exclude so briskly the possibilities of surgical excision in some of these benign strictures.

I will show two slides, and I would like to report a case which is different in that it is an advanced, long-standing stricture in a young lad 17 years of age. The injury was received at the age of 2½ years. He spent his whole life from that time until the age of 17 entirely on a liquid diet. However, he had lost a tremendous amount of weight and was having difficulty in school, and so, having been encouraged by our relatively low mortality figures in the carcinoma group, I decided to do a radical resection of the lower portion of his esophagus.

(Slide 1.) You will see that the new stricture begins rather high, and at operation I found that there was a tongue of ulcerated esophagus which extended upward, making it necessary to divide the esophagus opposite the left main bronchus.

(Slide 2.) The next slide merely shows the x-ray result. The anastomosis you see, is rather high here. Actually it was opposite the bronchus. That boy has done exceedingly well. Now, six or seven months following operation, he is eating a normal diet with his family. This is merely a suggestion of another way in which these cases can be handled.

I was intensely interested in Dr. Garlock's report. He has certainly covered the field most admirably in every possible respect. Inasmuch as the title of the article suggests that we ought to keep to the discussion of the causes of death after operation in these cases, I will confine my remarks to that subject and will quote our experience at the Massachusetts General Hospital. Using the Torek procedure there were fourteen cases with two deaths, which makes the mortality 14 per cent. One patient died of sepsis and the other of cerebral hemorrhage.

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*See page 383 for article by Adams and Hoover.

†See page 394 for article by Harrington and Moersch.

hepatic and glandular metastases, and an enterostomy for feeding purposes if so desired. At the second stage, done in one to two weeks, the carcinoma is resected and an esophagogastric anastomosis is performed between the proximal esophagus and the readily available gastric tube.

The one advantage in this procedure is that it allows surgical removal of carcinomas at a higher level in the esophagus with restoration of the esophagogastric continuity than could be obtained by the one-stage transpleural operation. Even though in rare instances the stomach has been brought as high as the arch of the aorta to allow anastomosis, the majority of patients with long-continued esophageal obstruction from carcinoma have a small stomach which prevents such a procedure.

A free jejunal tube can be employed in a similar manner if the stomach is found to be too small to fashion a gastric tube of adequate length. At the first stage a gastroenterostomy is performed between the stomach and a free jejunal tube. The free end of the jejunal tube is closed and placed within the chest during the first stage. The remainder of the operation is similar to this. Experimentally, it was found that more care must be exercised with jejunal tubes in order not to impair their blood supply than with gastric tubes.

If it is felt inadvisable to attempt intrathoracic anastomosis and an antethoracic subcutaneous Beck-Jianu gastrostomy tube is contemplated, considerably more distance can be obtained by resecting the lower costal cartilages and eliminating the necessity for the gastric tube to descend down and then over the costal arch. Further length can also be obtained by mobilizing the stomach at the esophagodiaphragmatic junction and bringing the stomach forward. If these suggestions are employed the gastric tube can easily reach the level of the neck. These procedures are largely based upon experimental observations and more work is necessary to determine whether or not they may become applicable.

DR. LEO ELOESSER, San Francisco, Calif.—I hesitate to prolong this long but profitable discussion. I should like merely to allude to one case of stricture of the esophagus that I treated a number of years ago through an open thoracotomy. The man was an ex-convict who had swallowed lye with suicidal intent, and he was sent to us by the otolaryngologists. He had been very effectually sounded, but no food got into his stomach. However, when I opened his abdomen I found that the sound had gone down retroperitoneally as far as the sacrum.

The sounding had been done for some months previous without very deleterious effects. In opening his chest, of course, I found a fistula. Proceeding from there I was able to get a large bougie up into his mouth. Also the hypertrophy of the lower segment was such as illustrated in Dr. Gurd's films of the gizzard. It was not too difficult to introduce a large bougie forcibly into his stomach from the upper esophageal segment.

It was easier, with the presence of this form of fistula, to re-establish continuity between his mouth and his stomach and gradually to dilate it. This man subsequently died because he pulled out the tube and we did not have him re-undergo the whole procedure.

DR. JOHN W. STRIEDER, Boston, Mass.—Relative to the causes of death in esophageal resection for carcinoma with primary esophagogastric anastomosis in the chest, I would like to mention briefly one experience which I had in a series of ten cases which I had an opportunity to follow completely. It also emphasizes the point which Colonel Bird made relative to the advisability of doing both esophagoscopy and bronchoscopy preoperatively. I believe this is an extremely important dual maneuver.

This patient, a woman aged 52 years, had a carcinoma of the esophagus discovered in a very early stage, 31 cm. from the superior alveolar ridge. The tumor itself was so small that in mobilizing the esophagus, although there was a positive preoperative biopsy, it was very difficult to palpate the tumor in the esophagus.

The tumor was resected and anastomosis was made after the suggestion of Dr. Garlock, by pulling the proximal esophagus anterior to the arch of the aorta. It was obvious from the second postoperative day that she was not aerating the left lung. On bronchoscopy, to our dismay, we found the left stem bronchus was completely occluded by extrabronchial compression.

This obstruction was known not to be due to tumor because of our preoperative bronchoscopy, and the observation was confirmed on subsequent bronchoscopy two days later. Because she had difficulty raising secretions and because even moderately copious secretions were of considerable embarrassment, in view of the fact that she was not using her left lung, we performed a tracheotomy in order to aspirate these secretions frequently and easily.

She died on the ninth day, and at autopsy we found there had been a leak in the anastomosis with a small localized abscess, perhaps the size of a half dollar and twice its thickness, in relation to the anastomosis, at the level of the arch of the aorta and also in relation to the left main stem bronchus. There was no general empyema. The left main stem bronchus was not incorporated in the wall of the abscess, rather the compression apparently resulted from the contiguous edema and pressure of the abscess.

I hesitate to mention our total experience at the Boston City Hospital. We have had an opportunity to explore twenty carcinomas of the esophagus, ten of which were operable. We have had two operative survivals and one living patient, over a period of two years. By way of partial extenuation I may say that we have dealt largely with the derelicts that one sees in a large municipal institution, and we have taken on all comers even though the chances of recovery seemed small.

DR. RALPH ADAMS, Boston, Mass.—I wish to show three additional cases of benign tumors of the esophagus. Illustrations appear in the article published in this issue of the JOURNAL.*

(Slide 1.) This was a woman 41 years of age, with rasping breathing, increasing for two months with no difficulty in swallowing. The mass moved upward and downward with the act of swallowing. On a statistical basis, substernal goiter would be by far the most likely diagnosis. The only other likely one would be an esophageal wall tumor.

The nature of this mass was not apparent until the recurrent nerve was demonstrated to run anterior to the tumor. In the course of some 23,000 thyroid operations it had never been observed at the Lahey Clinic that the nerve ran anterior to a thyroid tumor. Further dissection was made posteriorly. Some longitudinal esophageal fibers were found and a fibroma of the esophagus was removed.

(Slide 2.) This patient had difficulty in swallowing, of seven years' duration, but increasing only in the six months before admission. I would like to call attention to the characteristics of a benign intestinal lumen tumor as first brought out so clearly in our part of the country by Dr. Richard Schatzki. The barium fits over the top of the tumor somewhat as a cap. Barium flows freely around it and with fluoroscopy we saw the only place it did not flow was where the tumor was attached.

Directly opposite the tumor itself the esophageal wall looks normal, and one need only compare this slide with the following one with the narrow, tortuous channel created by carcinoma to see how markedly they differ in appearance. The tumor was too low to be removed through the neck and too large to be removed endoscopically. Its peculiar location in the organ in which it arose and its benign nature presented some specific problems in removal, and yet one must remove such a tumor with maintenance of esophageal continuity or accept all the hazards, cavity at the base from which the bronchiectatic lobe has been removed and this may not be completely obliterated for several days or weeks. This complete expansion of the remaining lobe should always be immediately possible providing the airway is cleared by the cooperation of an expert anesthetist. In spite of this our figures show that the incidence of collapse in our cases has been the same as in the 154 cases of Tudor Edwards and Price Thomas in which Gowar found collapse to have occurred fifty-one times.

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*See page 381.

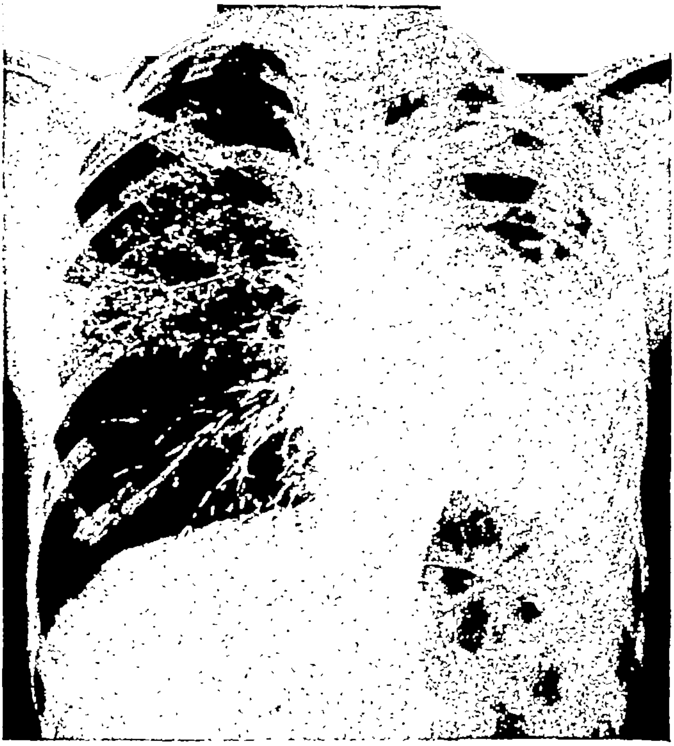

Fig. 1.—Roentgenogram showing cystic bronchiectasis which developed in the upper lobe following left lower lobectomy.

This secondary collapse has been attributed to several factors. Reflex bronchial spasm has been blamed, although the experiments of Band and Hall[3] failed to support this. Belsey[4] considered that swelling of the mucosa as a result of a recent bronchogram was a factor, while Tudor Edwards has expressed to us his view that inflammatory swelling around the stump is the cause of at least the more delayed cases. Many people have been struck by the better postoperative progress of patients whose pleural cavities were obliterated by adhesions. This has been explained by the retention of a certain amount of air in the remaining lobe so that it will be easier to cough the material out of the bronchi. This view led Bethune[5] to introduce his method of pleural poudrage. This method has been employed by us but as will be seen from our figures no reduction in the incidence of collapse was produced in the twenty-seven cases in which satisfactory pleural symphysis was obtained. We may state here that in spite of this we do still consider that pleural symphysis is an advantage and makes for easier convalescence and for easier treatment of collapse should it occur. It is felt that it is for these reasons rather than for its direct effect on preventing collapse that such authorities as Robinson,[6] Graham,[7] and Tudor Edwards[8] first noticed the more satisfactory course in patients whose pleural spaces were obliterated.

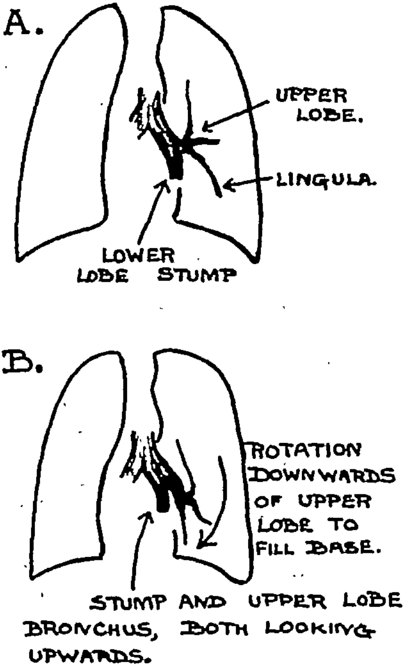
A.
UPPER LOBE.
LINGULA.
LOWER LOBE STUMP
B.
ROTATION DOWNWARDS OF UPPER LOBE TO FILL BASE.
STUMP AND UPPER LOBE BRONCHUS, BOTH LOOKING UPWARDS.

Fig. 2.—*A* represents the condition immediately after the lower lobe was removed. *B* shows what happened when the upper lobe expanded in an effort to fill the whole left side of the chest. The upper lobe bronchus rotated downward and came almost into line with the stump of the lower lobe.

In view of the unsatisfactory preventive measures previously employed and a strong feeling that patients develop collapse following a period of inability to cough effectively, it was decided to use bronchoscopy on all such patients immediately when the complication occurred. This procedure, which has been used over the past year, has proved most valuable therapeutically and has demonstrated some interesting facts about the etiology of the condition. First it is noted that not a single patient was seen in which the collapse was attributed to hilar swelling. Second, the collapse always appeared to be due to obstruction with sputum; third, when this sputum was removed re-expansion could be immediately obtained; and fourth, it was noted in all cases of lower lobe lobectomy that the upper lobe bronchus instead of coming off obliquely was looking almost directly upward. This means that it is nearly parallel with the stump of the lower lobe.

These findings have led us to believe that the course of events is as follows: The initial expansion of the upper lobe carries its bronchus downward in an effort to fill the cavity produced by the removal of the lower lobe. This brings the upper lobe bronchus almost parallel to the stump. During the first postoperative days pus collects in the stump from which it is difficult to be coughed up as there is no air behind to drive it out. If the stump fills it may easily spill over and block the upper lobe. It may still be coughed up, but, if the patient is not prepared to put up with the necessary pain, collapse will result. The symptoms will depend on the extent to which the smaller bronchi and bronchioles are filled and how grossly the obstructing material is infected. The deeper the pus gets into the lung the more permanent the condition will be. Among the various aspects of the case which this theory explains is the fact that collapse is more common on the left

TABLE I

Incidence of Postlobectomy Collapse (12 in 33 Cases or 36 Per Cent)*

				PER CENT
Onset:	From operation		1	8
	2nd to 5th day		9	75
	6th to 8th		1	8
	After 8th day		0	
	One case, exact time uncertain, during first 3 days		1	8
Side of operation:	Right	2 in 7		28
	Left	10 in 26		40
Lobes removed:	L.L.L.	2 in 13		15
	L.L.L. & lingula	8 in 13		62
	R.M.L.	0 in 2		—
	R.U.L.	0 in 1		—
	R.M.L. + R.L.L.	2 in 4		50
Age:	Under 16	5 in 8		70
	16 to 30	7 in 18		38
	Over 30	0 in 7		—
Amount of sputum:	Under 5 oz.	9 in 22		41
	Over 5 oz.	3 in 11		27
Drained:	Present	7 in 19		35
	Undrained	5 in 14		36
Adhesions:	Present	10 in 27		36
	Absent	2 in 6		33
Sex:	Males	5 in 19		20
	Females	7 in 14		50

*This small series was used as all the patients have received similar preoperative and postoperative care. Twenty-seven lobectomies were performed in the last 12 months.

than on the right side (17.5 to 42.5 per cent in Gowar's Brompton Hospital series). The left upper lobe bronchus normally comes off more obliquely than its opposite number on the right and thus after a left lower lobectomy the left upper lobe bronchus more easily comes into a parallel position to the stump than does the right upper lobe bronchus. This fact can easily be confirmed on bronchoscopy of these patients.

In practice it was found that only one bronchoscopy was required in adults, although recollapse may occur in children necessitating further instrumentation. It was found that even on the first or second postoperative day these patients stood bronchoscopy without being upset, in fact many felt immediately better. The procedure was always done in the theater under local anesthetic. By laying the patient on the good side the bronchoscope can be introduced through the side of the mouth into the affected lung with remarkable ease. In fact, it is our experience that it is easier to use the bronchoscope on these patients than on the usual run of patients. This is thought to be due to the greater ease with which they can relax.

TABLE II

TREATMENT AND RESULTS

CASE	ONSET (DAY)	BRONCHOSCOPY	IMMEDIATE RESULT	AGE (YR.)	FINAL RESULT
1	5th	+	Expanded	31	Good
2	6th	+	Collapsed until 10th day	7	Good
3	3rd	+ × 2	Expanded	14	Good
4	?	+	Collapsed until 5th week	6	Good
5	1st	+ × 3	Expanded	14	Good
6	2nd	+	Expanded	21	Good
7	4th	+	Expanded	23	Good
8	3rd	+	Expanded	31	Good
9	8th	+	Expanded	20	Good
10	5th	–	Collapsed until 9th week	18	Died, C.A.
11	3rd	–	Collapsed until 5th week	9	Good
12	4th	–	Collapsed until 12th day	21	Good

An interesting point arose in an early case in which drainage was not carried out. The patient developed collapse that was thought to be due to the presence of fluid in the pleural cavity. Aspiration of 13 ounces resulted in only the slightest improvement in the radiographic appearance and it was realized that this had been a dangerous move since the increase in intrapleural pressure with the bronchus plugged with pus would only tend to draw that pus further into the smaller bronchi. Bronchoscopy was therefore immediately used after which, with the bronchi clear, a further aspiration resulted in complete re-expansion. The same situation has since occurred in another case but here bronchoscopy was immediately undertaken.

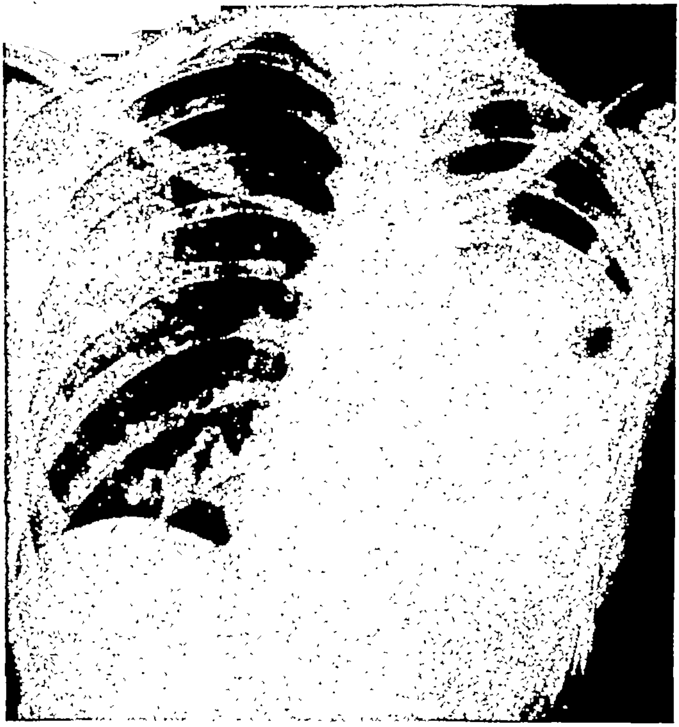

Fig. 3.—Radiogram of patient in whom drainage was not carried out. The left upper lobe is collapsed with some fluid and air in the pleural space.

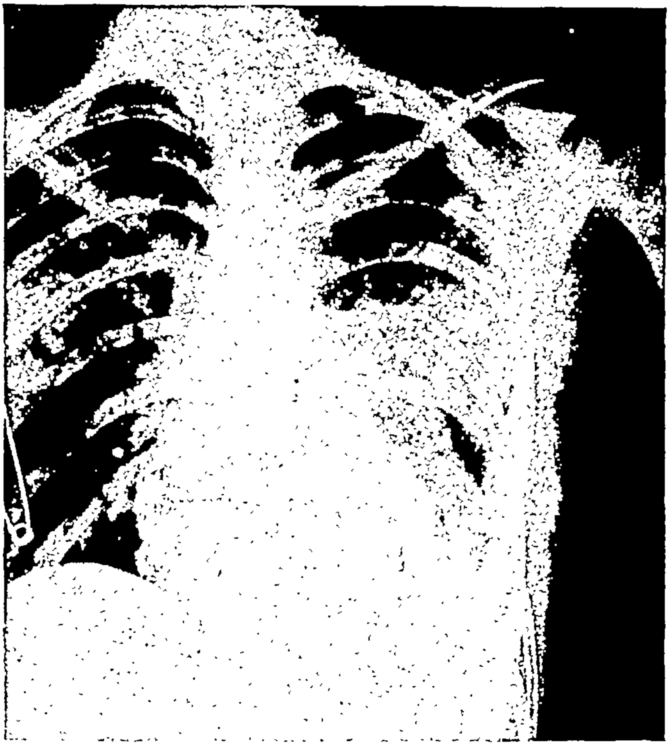

Fig. 4.—Radiogram of same patient and made same day as Fig. 3. Bronchoscopy was performed followed immediately by aspiration of the chest, and complete re-expansion of the left upper lobe was obtained.

These findings with regard to the part played by the material collecting in the hilar stump have led naturally to methods being adopted to prevent collapse by keeping this stump dry. It appeared that this should be possible if early postural drainage was employed by laying the patient over on his good side. It is too early to speak dogmatically about this method but one word of warning may be of assistance. It has been found that such posturing if employed between the seventh and twenty-first days is likely to precipitate a bronchial fistula; however, it would appear that posturing between the second and fifth days is without danger and gives hopeful signs of being useful. It is to be noted that in our series 75 per cent of the cases of lobar collapse occurred in this period between the second and the fifth day. This postural treatment is made much easier in view of the fact that drainage is usually not carried out in our cases.

SUMMARY

1. From several aspects postlobectomy lobar collapse is a serious complication.

2. It is due to the obstruction of the bronchi with pus in association with certain mechanical alterations in the shape of the bronchial tree after lobectomy.

3. The longer this pus is left plugging the bronchi, the deeper it will pass into the lung and the more permanent will be the condition.

4. Once collapse occurs it should be treated immediately by bronchoscopic aspiration, the results of which are highly satisfactory.

5. Postural measures give rise to some hope of reducing the incidence of the condition.

We wish to thank Mr. W. J. Pardoe of the Anatomy Department of the Birmingham University for his excellent photographic work which was made possible by the courtesy of Dr. C. F. V. Smout, acting professor.

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FLAP VALVE CONSTRUCTED FROM A MEDICINE DROPPER

ESKE WINDSBERG, M.D.
PROVIDENCE, R. I.

A ONE-WAY flap valve can be constructed easily from the rubber bulb of the ordinary medicine dropper. The closed end of the bulb is cut with scissors to form a fish mouth. This makes a vent through which air under pressure will travel one way only. A bulb syringe attached to the normally open end of the medicine dropper bulb can be made to demonstrate this fact (Fig. 1, *A* and *B*).

When the rubber bulb at hand is rather thick or hard, a large fish mouth is required. Conversely, when the rubber is thin and soft, a smaller fish mouth will work better. An elastic band may be placed around the bulb at the base of the fish mouth to regulate the force required to open the vent, and to insure its closure.

The application of this bulb valve is as simple as its construction. Through the medium of the glass part of the dropper it is attached to the end of the intrapleural catheter. (The bottleneck end of the glass part should be cut away.)

At the termination of an operation the flap valve is attached to the chest catheter and at once incorporated into the wound dressings. Except in unusual circumstances such as hemorrhage, it needs no further care or even inspection for at least twenty-four hours. The patient is protected against excessive intrathoracic pressure, and expansion of the remaining lobes is automatically promoted. Should a leak occur in a bronchial stump, the vent will function automatically to protect the patient. Liquid drainage, free from clots and fibrin, will also come through.

The intrathoracic status can be tested by unhooking the flap valve and attaching a bulb syringe. Usually this is not required until the day after operation. The advisability of continuing the use of the valve in the dressing, of using an extension tubing and placing the valve at the end into a bottle, or of discontinuing the use of the valve can then be determined. If excess negative pressure is feared, a mercury safety vent can be incorporated into the system.

I have found this simple device handy and useful on many occasions following lobectomy and pneumonectomy. It is being used routinely, incorporated in the original dressing in the operating room. This enables safe handling and transfer of the patient to his bed. It pre-

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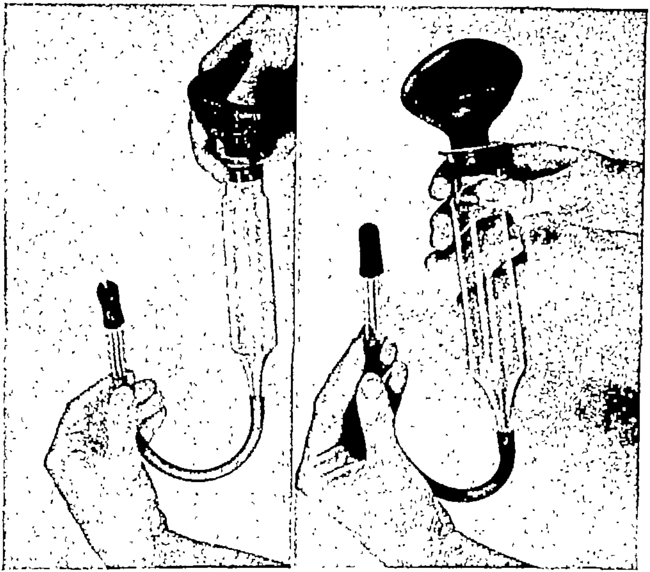
From the Rhode Island Hospital II Surgical Service, and the Thoracic Clinic.

Received for publication May 31, 1944.

vents any sudden increase in intrapleural pressure during this critical interval and removes chance extraction of the newly placed chest catheter. For patients in whom drainage is a primary necessity, especially in the presence of fibrin, it is usually not to be employed, since it will not work. However, during the first day or two following the drainage of a massive empyema, the device on occasion has proved desirable in conjunction with underwater drainage. Its use here minimizes the mediastinal shift due to respiration.



A. *B.*

Fig. 1.—*A*, Pressure being applied forcing air out through fish mouth of rubber bulb. *B*, Rubber of bulb syringe remains collapsed with fish mouth closed preventing any air from returning to system after thumb pressure has been removed.

The described device is superior to the finger-cot or to the finger part of a rubber glove into which a fish mouth is cut. Not every finger-cot preparation will work, and one which is found to work at the outset cannot always be depended upon to work continuously. The finger-cot preparation works well only when hanging freely in an open bottle, out of water. The bulb fish mouth functions equally as well under water as it does above water. Simple underwater drainage may allow wide shifting of the mediastinum during respiration or coughing.

The outstanding feature of the bulb fish mouth preparation is that it also works well when incorporated into the chest wall dressings. This

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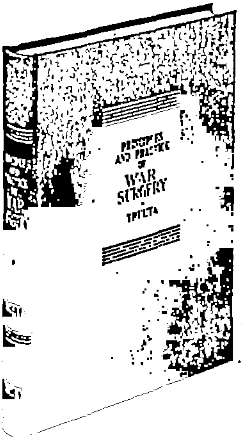

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